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Southern Medical Journal

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Southern Medical Association, Thirty-Fourth Annual Meeting, Louisville, November 12-15, 1940

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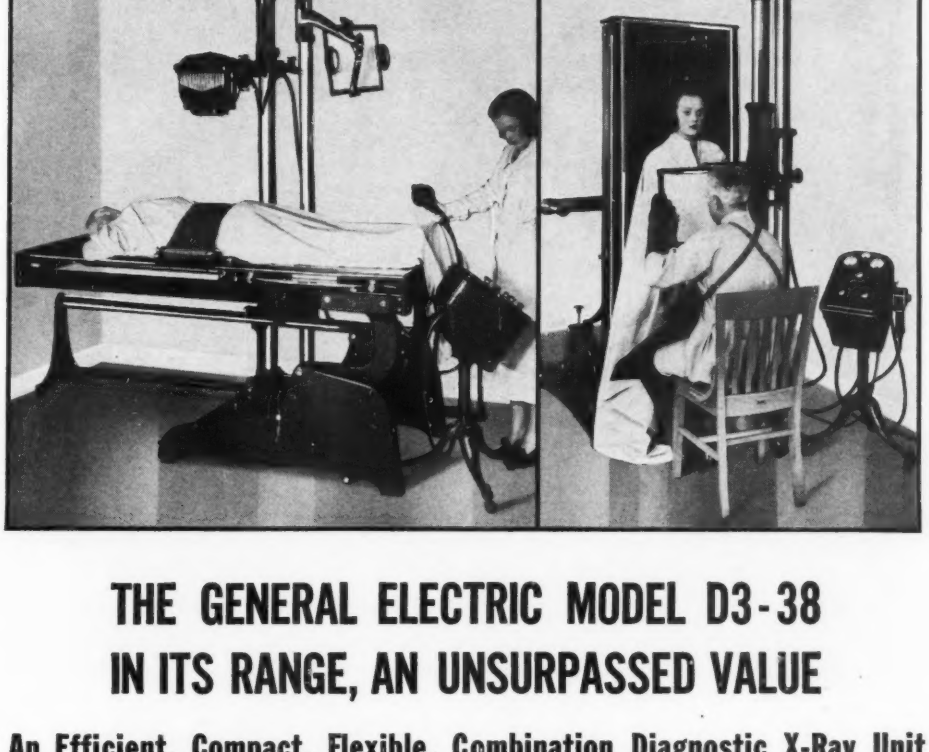

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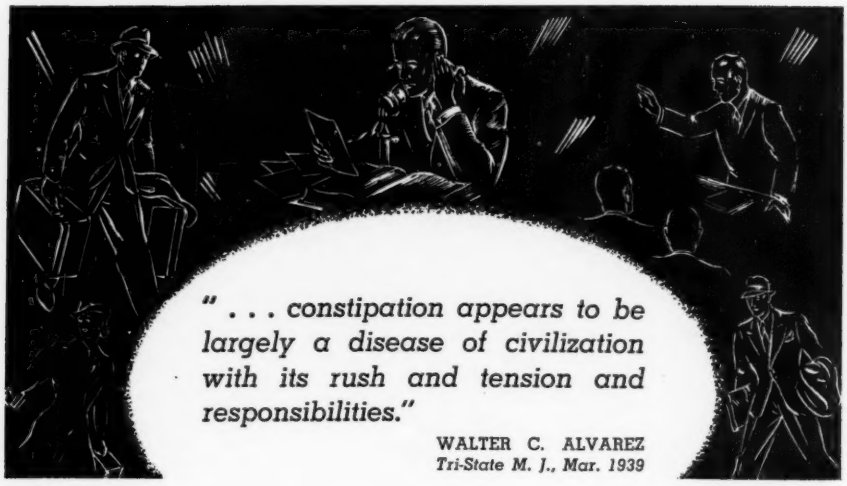
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WALTER C. ALVAREZ
Tri-State M. J., Mar. 1939

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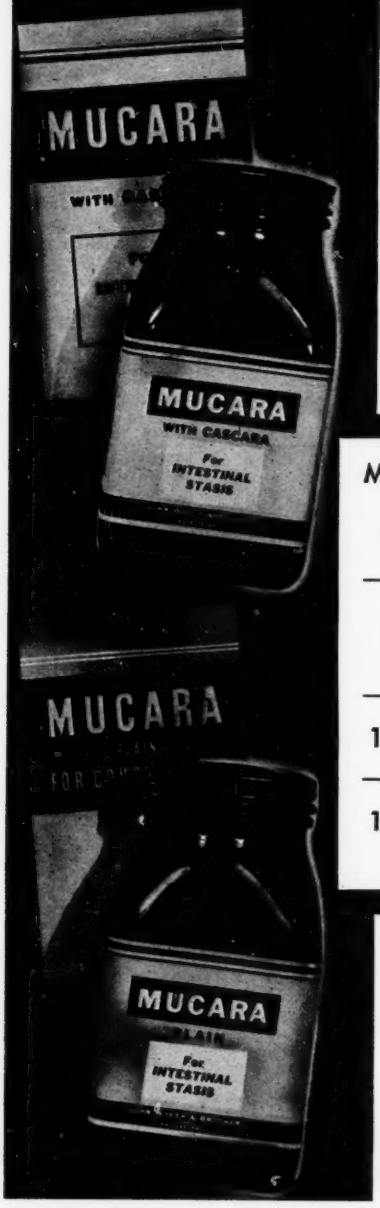

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* American Journal Digestive Diseases, 5, 5, 315, July, 1938

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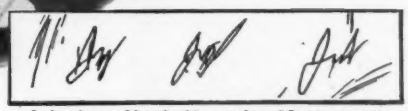
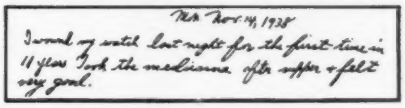


1. Specimen of handwriting on day of first treatment



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1. J. Mt. Sinai Hosp., 6:93-99, July-Aug., '39
2. Pennsylvania M. J., 43:67-69, Oct., '39

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"Proceedings of the Society for Experimental Biology and Medicine", 40:157, 1939.

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*Unpublished data—JOSLIN, C. L. and CHRISTENSEN, T. A.

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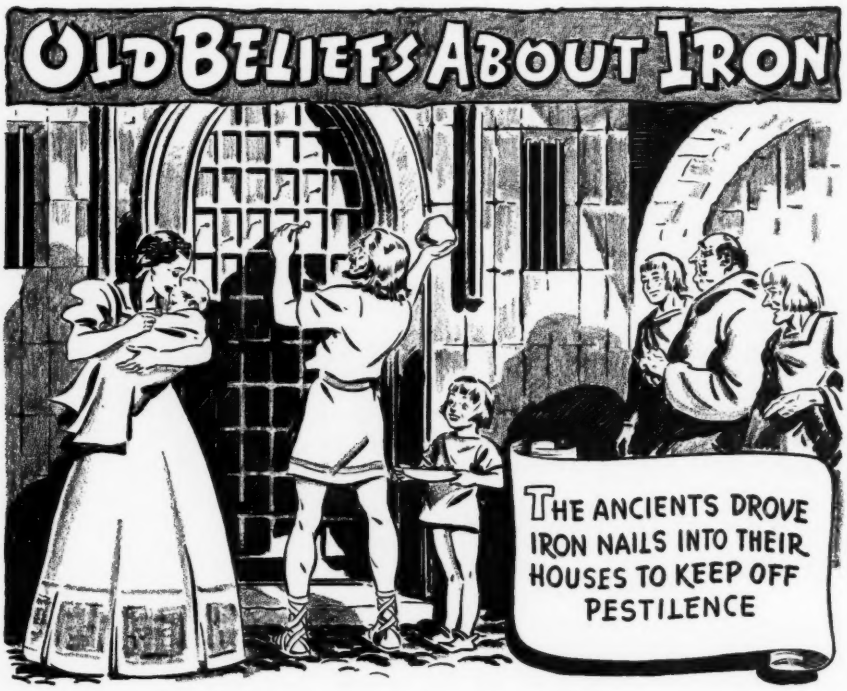

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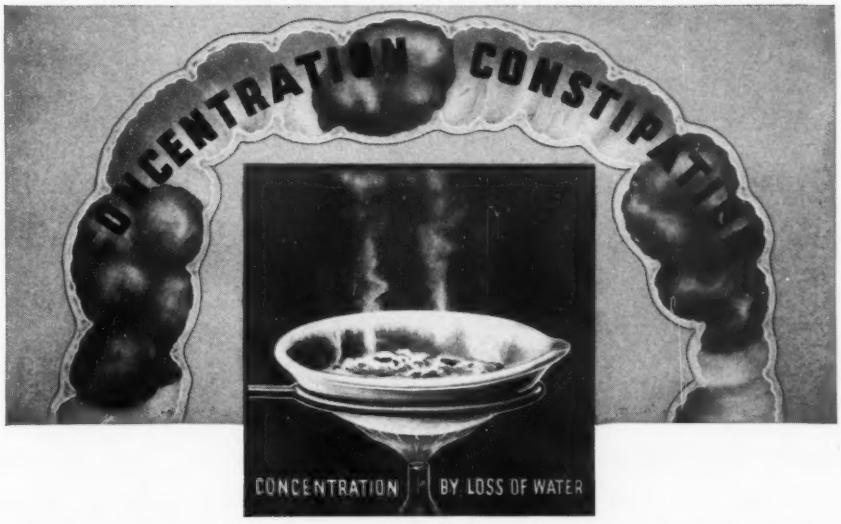

When the Concentrating Function of the Colon is too Efficient

Typical of the obstinate case of constipation is accentuation of water deficiency (dehydration)—"the condenser-like function of the colon is too efficient and the feces become too concentrated and small in bulk to act as a proper stimulus to defecation."*

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*Welch, P. B., and Kauders, F. H.: The Physiologic Approach to the Correction of Constipation, South. M. J. 31:709 (July) 1938.

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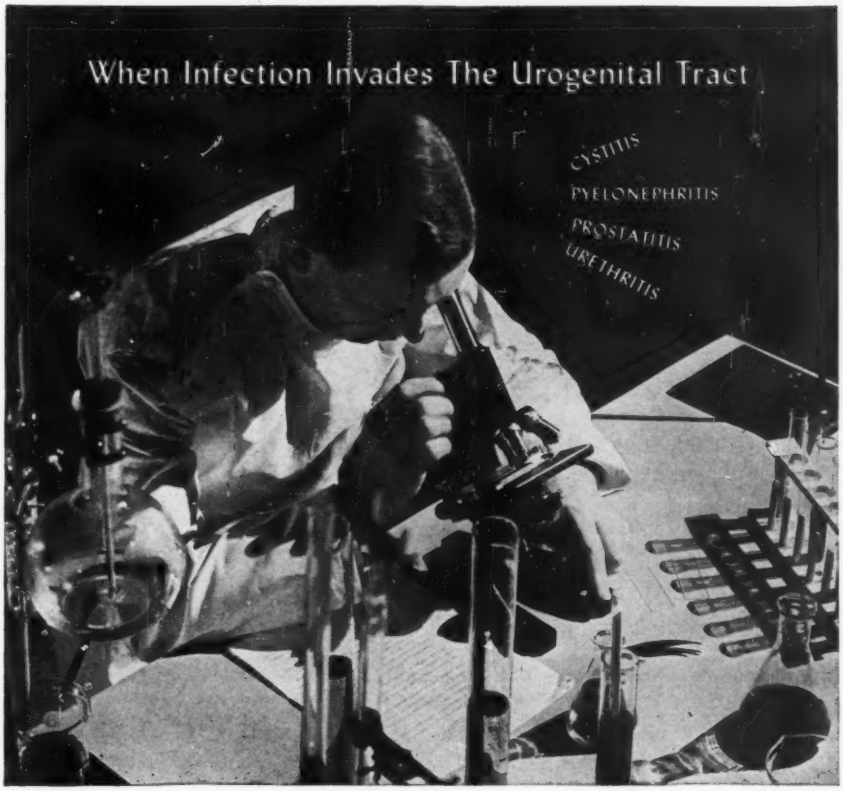

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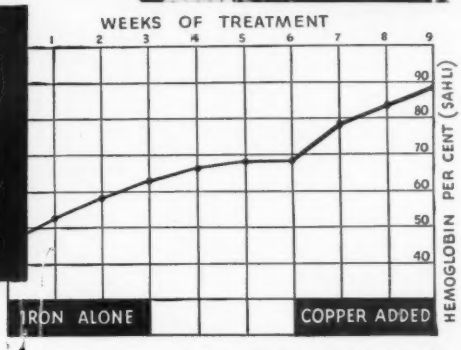

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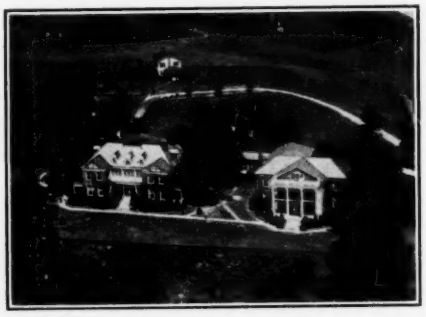

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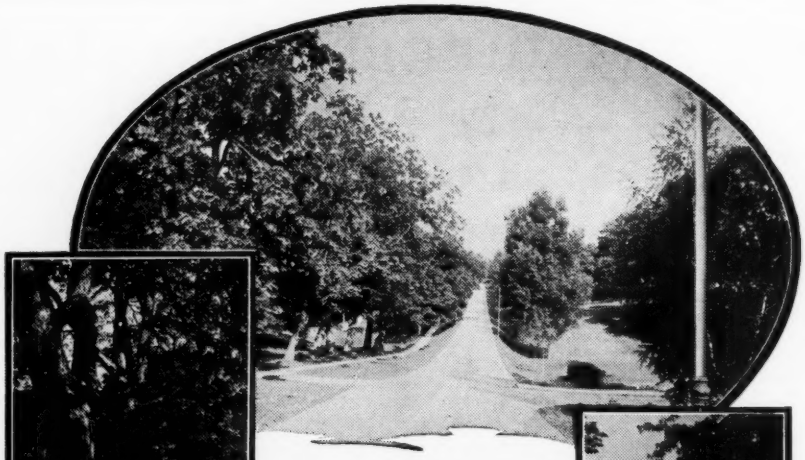

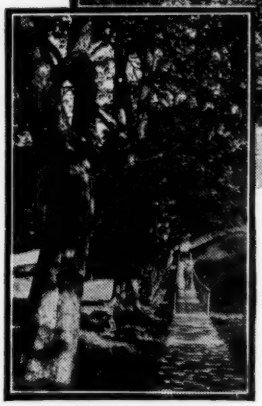

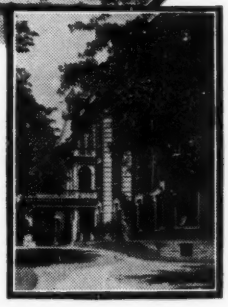

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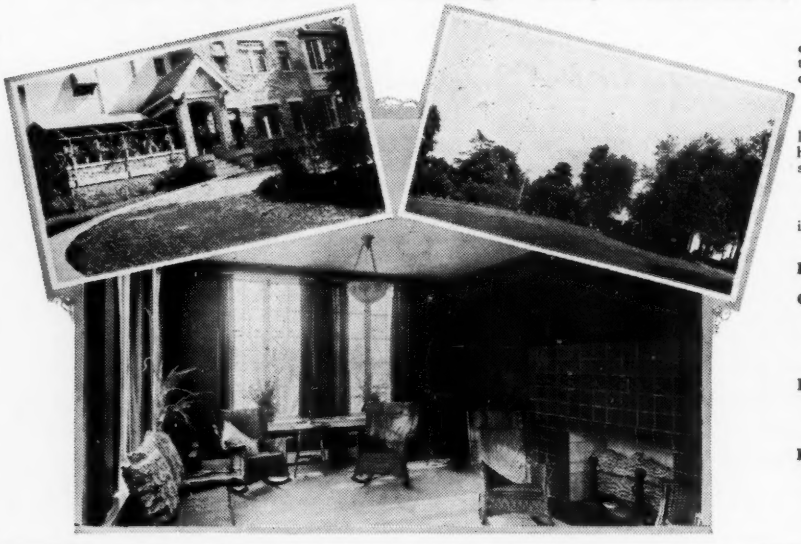

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Volume 32 DECEMBER, 1939 Number 12

ADVANCES IN THE TREATMENT OF EVERYDAY WOUNDS*

By BEVERLY DOUGLAS, M.D.
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In spite of all the contributions that have been made to the subject, a great many points concerning the treatment of wounds still need attention. Many facts rather commonplace to the reconstructive surgeon need to be passed on to the general surgeon who is responsible for the treatment of the great majority of wounds.

Definition.—In brief general terms, a wound may be defined as a division of the continuity of tissue by physical or chemical action. This is understood to include defects in tissues wrought by circulatory deficiency and by the multiplication of bacteria and neoplastic cells.

Not only war and industry, but acts of every day living, as well as many forms of medical therapy, such as x-ray, expose individuals to hazards which may produce wounds.

Up to the time of Ambroise Paré's contributions, the treatment of wounds was obviously a hit and miss affair. After Paracelsus gave forth his dictum, "'Tis nature healeth wounds, not meddling," physicians very generally adopted this teaching. True enough, Paré's most famous saying, "Je pensay et Dieu la guarist" (old French for "I dressed his wound, God healed him") might seem to imply that he agreed with Paracelsus, yet his whole professional career is filled with acts which show his deep conviction that man, while maintaining an humble reverence toward God, should take an active part whenever possible in assisting the natural processes of the body toward healing. As will be shown by quotations from Paré's work, he was obviously the first in the world's history to stress the importance of the scientific approach to wound treatment. Part of this was due to an accidental circumstance, namely, his lack of boiling oil to pour in deep gunshot wounds of soldiers, but his work on suture and on the importance of the general hygiene and mental status of the wounded patient bears all the marks of a true investigator. It is an important though little known fact that he kept the record of the healing of ulcers through tracings on paper. As far as I have been able to learn, this was the first quantitative work to have been done on wounds in the world's history. His work marks the beginning of the modern scientific approach to the subject of wound healing and treatment. The ideal which I feel should be aimed at in wound treatment is so to increase our knowledge of all the factors concerned that our methods will be based upon the fullness rather than the leanness of our learning as we apply our experience to individual cases. Paré contributed richly to the fulfillment of this ideal.

The modern concept of the operative treatment of wounds and deformities should be based upon a consideration of both form and function. At the end of a harelip operation a beautiful anatomical result will usually satisfy the family and elicit their gratitude. Unless the lip appears too long at the suture line at the end of the operation, a short or a notched lip will inevitably result later on. Wardill, of Newcastle on Tyne, has likewise beautifully demonstrated the futility of a nice anatomical result on a cleft palate without functioning muscles which will allow the patient to speak properly.

Classification of Wounds.—Little profit can come from attempting to classify wounds in a paper of this length. The division of wounds into (Type 1) small wounds or large superficial ones, and (Type 2) extensive division of tissues with loss of tissue and possible damage to important structures, including (a) those which may be sutured directly, and (b) those which require grafting or shifting of flaps for closure will answer all practical purposes. It must be admitted without contention that the average wound is actually a mixture of many varieties. We rarely see a laceration without some contusion of the edges. The definitions of the various varieties of wounds are too well known to occupy space here. They will be taken for granted as a few points in treatment are emphasized.

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Early Treatment.—(A) *Abrasions.* The principle here should be to obtain rest for the abraded and neighboring tissues. Since sensitive nerve endings are usually exposed over a greater or less surface area, they should be protected. If gauze is used, it should be moistened with saline and the moisture retained by placing over the gauze an occlusive layer of impermeable material such as oiled muslin or cellophane. This cannot always be done in an ambulatory case. It is, therefore, important to apply a fairly heavy coating of a bland ointment and to dress the area infrequently. Often just the opposite is done. I have many times been urged by the patient and the family and even by interns to dress such a patient every day. By so doing nothing is to be gained and we have the definite danger of introducing infection and tearing away delicate growing tissue as well as that of causing further pain to the patient. Dressings three days apart should be the rule unless definite infection supervenes. In most cases the latter may be avoided by immediate careful cleansing and sterilization of the skin. The raw surface needs no sterilization except in those instances in which there has been an unusual degree of contamination. In most abrasions, careful cleansing of the skin followed by a firm strapping with elastic adhesive directly on the surface will result in rapid growth of the basal layers. In many instances this has resulted in much comfort to the patient and the rapid healing of the skin; in fact, when the plaster is removed at the sixth or seventh day, the skin may not only be healed but a layer of desquamated cells may be attached to the adhesive.

(B) *Incisions and Lacerations.*—The same principles are concerned in the closure of small wounds of these types as with the large. Complete details of closure should, therefore, be sought under Type 2 or extensive wounds which are discussed later. Clean incised wounds should be closed layer by layer to prevent dead spaces. Since no tissue loss is involved, no tension sutures are necessary. If closure must be delayed for any reason beyond six hours after injury, incisions should be loosely closed with drainage or packed open and closed secondarily. In the case of lacerations, closure should be performed only after débridement. Tension may be avoided by undercutting, by relaxation incisions or by other plastic methods. Stay sutures tied over rubber should be employed to hold the wound edges in snug, but not tight approximation.

(C) *Contusions.*—Ice packs should be applied as an immediate measure to contusions of the skin and subcutaneous tissue. This may reduce symptoms and delay the onset of inflammation. In those cases complicated by hematoma formation, blood should be withdrawn through a needle under strict aseptic precautions and the part strapped firmly with elastic adhesive or otherwise bandaged to prevent recurrence. Should there be recurrence, it may be necessary to locate and tie the offending vessel. If, later on, clotted blood is found to cause inconvenience or deformity, it should be pressed out through a small incision and strapping applied. In all cases the contused part should be put at rest by proper splinting and a pressure bandage.

(D) *Avulsions.*—I wish only to mention two practical points in connection with avulsed wounds. The first of these is to urge surgeons to replace small portions of the body such as the ends of fingers, parts of the ears and nose. I have quite a large series in which success has followed such replacements after careful washing with salt solution, trimming away of dirt and surgical preparation of the skin as would usually be done if the part were still attached. It is to be understood that such replacements or replantations should be undertaken only if the part involved is fairly small or is supplied by a terminal circulation; in other words, parts of the body such as the finger or toe or an ear. I am happy to report that success has followed an extremely high percentage of such replacements in my cases. The second point relates to avulsions of large masses of tissue, notably avulsions of the scalp which may be attached by only a narrow bridge of tissue. If the customary tests show any blood circulation to be present, replantation in a loose manner without tension on sutures may result favorably. If not, it should not be attempted. There is one other recourse. This is to prepare the part properly and to cut small deep ("pinch") skin grafts from the detached skin, including the hair follicles, and to place them fairly closely on the denuded surface.

(E) *Puncture Wounds.*—Since these wounds are made with a narrow instrument or missile which may penetrate to any depth and damage important and even vital structures, the rule should be to "look before we leap." The immediate treatment should be to splint the part and not to probe or to pull out a sharp weapon until the patient is in the operating room and is pre-

pared for a major procedure. The natural reaction is to try to remove the foreign body at once. This involves probing which may introduce further infection into the depths of the wound. Another impulse is to pull a long object such as a dagger or a pencil out immediately. There is no telling how many lives are lost each year through just such an impulsive action. I may emphasize the importance of not doing this by a few cases.

The first was a stab wound of the chest inflicted by a Negro man on a student. The postmortem findings indicated that death resulted not from hemorrhage, but from pneumothorax. If the knife had not been withdrawn, or if after withdrawal it had been possible to press the air out immediately and to temporarily seal the wound edges together, it is almost certain that the student would not have died. The other patient, it so happened, was my own son, a boy of twelve years, who had the misfortune to fall on a long, sharp lead pencil which he had stuck, point upward, in the left hand pocket of his trousers. The point perforated upward through the abdominal wall, grazing and tearing the peritoneum. It finally perforated the diaphragm and pleura and lodged against the left lung base. I happened to be with him, and immediately held his body rigid. With the aid of some of his playmates, he was placed prone on the back seat of an automobile and was taken to a hospital. After visualization of the pencil by x-ray, he was operated upon by Dr. Brooks, who followed the pencil upward to its point. By having him prepared for a major operation, protection against abdominal complications resulted. The wound in the pleura was repaired, although a definite small pneumothorax was visualized after the operation. The abdominal wound was drained by a rubber tissue slip extending to but not through the pleura. This was removed in 48 hours and *per primam* healing resulted.

It is my practice in the Out-Patient Department to localize all small foreign bodies such as needles and bullets under the fluoroscope, to introduce a few drops of procaine hydrochloride into the skin, push the needle in until it touches the foreign body, and, still visualizing it, cut down upon it and lift it out with forceps. This fairly simple procedure will result many times in a great saving of time to the staff and of pain to the patient.

(F) *Incisions and Lacerations.*—Unless very small, such wounds will need to be sutured. The principles as well as the details of technic involved in their closure are described under "Type 2" wounds.

Management of Type 2 Wounds.—These include wounds involving extensive deep division of tissues or their loss, as well as those in which there is crushing of muscle, a compound fracture or various combinations of superficial and deep lesions. It is almost trite to say that the first considerations will be the condition of the patient. Yet how often this is overlooked! First aid, namely, splinting adjacent joints and control of bleeding by tourniquet or other pressure methods, along with avoidance of rough handling of the patient in transport to the hospital must not be neglected. We do wish to emphasize that no further measures than these should be carried out before the patient has been aided to react from shock. Through appropriate measures, at least two of the three kinds of shock which have been clearly defined through the experiments of Dr. Blalock may be produced by wounds. These are (a) shock from hematogenic and (b) shock from neurogenic factors. Their treatments are different and should be carefully considered. To the hematogenic variety which is marked by an early diminution in blood volume with peripheral vasoconstriction he attributes hemorrhage or extensive trauma to muscles, with plasma loss into the tissues as the typical causes. The blood pressure declines more slowly and later than in shock of the primary neurogenic variety. Early transfusion of whole blood without injection of vasoconstrictor drugs is indicated. If the blood pressure is allowed to remain low for several hours, even transfusion will usually fail to save the patient.

The second or neurogenic type is that which results from trauma to the central nervous system directly or reflexly from a blow on the abdomen. The lowering of the blood pressure in spinal anesthesia affords another example. Here there is an early decline in pressure due to loss of constrictor tone in the vessels. In this type Blalock feels that the administration of vasoconstrictor drugs with, later, blood transfusion or other fluids, if necessary, is to be advised. I quote from his article:

"In the case of severe injury when the diagnosis has been made and in which damage to the brain is not suspected, morphine should be given. In the presence of a gross injury to an extremity the application of a tourniquet is usually the quickest way of stopping hemorrhage. However, it is very important that the tourniquet should not be left in place for a considerable time if an attempt is to be made to save the part. In the presence of a low or declining blood pressure, local or gas-oxygen anesthesia is preferable to ether or chloroform or spinal."

The Treatment of the Wound Proper.—I shall not attempt to discuss wounds of special regions such as penetrating wounds of the abdomen, pleura or deep wounds of the face. In extensive facial wounds the immediate calling in of

a plastic surgeon instead of waiting may shorten the usually long, tedious aftermath of surgical procedures. By carefully planning flaps to swing into areas of lost tissue on the face as soon as the injury occurs, the seemingly impossible may be accomplished with relative ease. Such procedures are commonplace to the skilled reconstructive surgeon. The specialist should have a greater part in emergency procedures than he is customarily given at the present time.

For practical purposes of treatment we divide deep wounds further into: (a) those which are deep and extensive but which may be sutured primarily and (b) those involving loss of tissue extensive enough to require grafts or flaps. We are not particularly interested in describing exact procedures for the treatment of either class. The surgeon must individualize each wound and treat it according to his best judgment. We are, however, deeply interested in the principles, a knowledge of which will enable him to interpret his experience and to act according to the best dictates of his reason. In the past there has been much misapplication of methods in the treatment of certain wounds. I shall shortly bring this out in the discussion of antiseptics.

The principles which I shall discuss for the treatment of the second division of wounds, to wit, lacerations with or without loss of tissue and their complications, are applicable in general to all forms of wounds and wound healing. The actual carrying out of treatment will be quite different according to whether the wound is situated on one of the extremities or on some other portion of the body. The reasons for this are that a limb lends itself to its covering by a jacket of a type I shall shortly describe and likewise it lends itself to the control of hemorrhage by tourniquet and to enforced rest by splinting. If then I discuss principally wounds of the extremities, it is because the general principles applicable to them are also applicable to wounds of other special regions. Actually most wounds occur on some portion of the extremities.

Turning then first to wounds involving too large and too deep a loss of tissue to be closed by direct approximation, it should be kept in mind that any plastic procedure directed toward swinging in or transferring tissue to the site of loss will, if successful, be tremendously time-saving and possibly life-saving to the patient. Such a procedure in skilled hands will not necessarily prolong the length of operation or the hemorrhage and resultant shock therefrom. If, however, the patient is in a severe condition when brought in, as was the one shown in *Fig. 1*, no procedure, not even the simplest, should be carried out until shock has been combated. If a fairly simple plastic procedure will not suffice to allow of suture without tension, any flap of tissues which is available should be tacked down near but not to the opposite edge, thus preserving its usefulness and making further closure easier. Reid[1] sums up the situation in these words:

"An avulsed flap of skin is far more useful alive and unsutured than dead from the anemia of the tension of the sutures which place it beautifully back into perfect position. To tack a flap of skin loosely back one-half inch or more from the opposing edge is, I think, the hardest things in the world for a young surgeon to do."

This principle is applicable throughout general as well as plastic surgery. Débridement* or the cutting away of dead and devitalized tissue will often produce a wound whose edges it may not be possible to approximate without tension. It is a notable fact that the surgeons of the World War who obtained the best results were those who approximated wounds loosely and with few sutures. Most of the others soon learned their lesson. In case deep wounds cannot be filled in by flaps of skin and deeper tissue without producing tension, we believe that if it is at all possible, one should immediately apply a skin graft from a distant part (*Fig. 4*). In case such a wound is infected, either a "pinch" or a perforated "sieve" graft will offer the surest chance of a take. In relatively uncontaminated

*Epluchage is the more proper term.

Fig. 1

Full thickness loss of two-thirds of lower lip from fall against barbed wire. Shock too great for immediate repair. Reconstruction accomplished in one stage by swinging pedicle flap of skin and subcutaneous tissue from right cheek above upper lip to remaining shoulder of lower lip and lining it with flap of buccal mucosa from inside right cheek. If immediate repair could have been done the patient would have been spared much suffering and loss of time.

Fig. 2
Skin grafting by pattern without the employment of sutures. (Author's case, *Annals of Surgery*, 1923.)

fields other forms of grafts may be used. It is interesting to note that the observations and experiments of Reid, Carter, the writer and others demonstrate that when a fairly deep cavity is grafted, instead of being filled up with a flap, a gradual leveling up of the grafted area with the surrounding skin occurs during a period of months following the operation. A general consideration of methods of skin preparation or sterilization of the technic of suturing, and of the nature of the dressing will fall more properly under a discussion of wounds in the next heading.

Treatment of Wounds Which Are Deep and Extensive but Which May Be Primarily Sutured.—All deep wounds are potentially contaminated. The degree of contamination should determine the method of repair employed. Success will depend upon the nicety of judgment possessed by the operator.

Skin Sterilization.—For an accurate discussion of this subject we refer the reader to Homans'[2] "A Textbook of Surgery." It is my belief that after shaving with lather a wet technic should be carried out throughout in contaminated and dirty wounds. This may be done by thoroughly irrigating the wound tissues and skin with 1-3,000 bichloride of mercury solution followed by an irrigation with warm normal saline to check the corrosive action of the bichloride. No iodine should then be used. In relatively cleaner wounds the skin edges may be shaved dry, then painted with iodine, up to but not including the raw surface. The latter, if cleansing is needed, may be cleaned gently of dirt and blood clot with a small gauze swab soaked with ether, but not saturated to the dripping point. On badly soiled skin, soap and water may precede the bichloride, but no "wet" technic should ever precede iodine.

Débridement.—The principle of cutting away all dead and devitalized tissue, while practiced before the World War, was reemphasized in the Carrel-Dakin technic for the treatment of infected wounds. It aims at converting the badly contused and lacerated edges of the wound into clean healthy tissues which will heal *per primam.* It emphasizes the importance of good blood supply as a *sine qua non* of rapid healing. After exploring the depths of the wound for foreign bodies and divided tendons, large vessels and nerves and suturing them, the wound edges should be sutured in layers so as to leave no dead space.

PRINCIPLES INVOLVED IN WOUND CLOSURE

Contact.—Throughout the remainder of the discussion I shall emphasize contact of wounded tissues in proper relationship as being extremely important in wound healing. Without contact, even though a graft misses the surface which it is intended to cover by a mere millimeter, little possibility of success may be expected. When wound edges are not in contact, one may predict that the dead space between them will fill up with blood clot, that further hemorrhage may occur and that infection may also ensue. We are speaking, of course, of wounds in which no drainage is thought to be indicated. In the latter, as has been stated, loose approximation will be

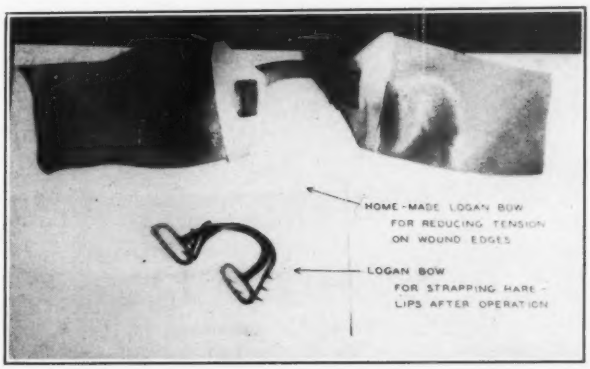

Fig. 3
Use of Logan bow principle in fixation of wound edges.

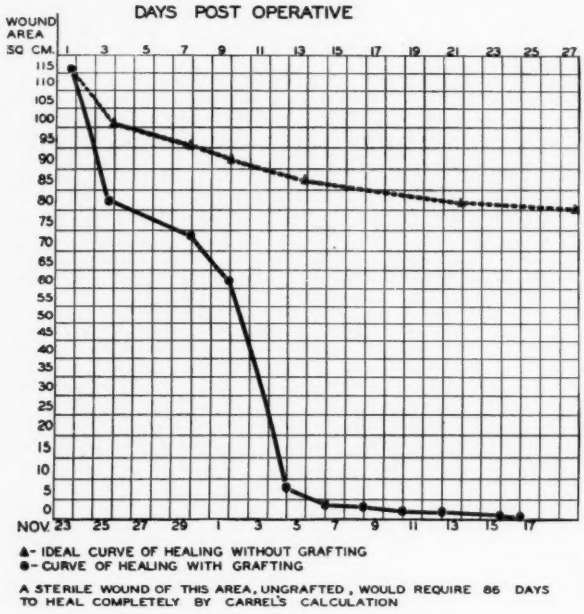
DAYS POST OPERATIVE
WOUND AREA SQ CM.
NOV
▲- IDEAL CURVE OF HEALING WITHOUT GRAFTING
●- CURVE OF HEALING WITH GRAFTING
A STERILE WOUND OF THIS AREA, UNGRAFTED, WOULD REQUIRE 86 DAYS TO HEAL COMPLETELY BY CARREL'S CALCULATION

Fig. 4
A sterile wound of this area, ungrafted, would require eighty-six days to heal completely by Carrel's calculation.

desirable and the insertion of drains at certain points will greatly favor healing. In emphasizing contact of clean wound edges as being all-important, I do not wish to imply for one moment that the contact should be anything but light. In order to avoid more than slight pressure on wound edges the horse hair technic as extensively used by Blair and the recent trial use by Gillies of fine elastic rubber suture material both demonstrate the principle of maintaining light but constant contact of the wound edges. Halsted[3] always emphasized the utter futility of tying sutures which approximate skin edges too tightly.

Suture Material.—The best results in wound healing will be attained when the surgeon realizes that the least suture material that can be employed in keeping with the proper attainment and maintenance of light contact is the best. The plastic surgeon realizes that he must not place any ties under a skin graft if he can possibly avoid them. To use such a technic presupposes the control of hemorrhage through the employment of heat and pressure locally at operation and some form of pressure dressing over the graft afterward. I must confess that the suture material which I find it necessary in most cases to employ at the edges of skin grafts causes a delay in healing out of proportion to the take of the center of the graft. This is almost certainly due to the suture's acting as foreign bodies at the weakest part of the graft, namely, the periphery, at which a division of the skin has already been made. *Fig. 2* shows one of a series of small patterned Thiersch grafts which I applied with success to the ears of several severely burned patients at New Haven.[4] No sutures were used to hold the grafts on. Crosswise strips of adhesive, previously sterilized, held them in position until takes occurred. As soon as the plasma which collected under them had been drawn out with a needle, the whole surface was sprayed with a paraffin rosin preparation. *Fig. 3* illustrates the use of home-made Logan bows which we have successfully employed to hold the edges of lacerated wounds together which are too contaminated to suture or which because of infection have had the edges pulled apart, due to the cutting of the sutures.

Halsted first emphasized the disadvantage of burying large ties in his use of fine silk for ligatures. Whipple has recently shown that a catgut tie of equal strength to fine silk will create a foreign body reaction approximately twenty times as great. This fact is of great importance in plastic and thyroid surgery. Meleney[5] gives many reasons for the superiority of silk over catgut. Chief among these are that it is almost inert in the tissues, while catgut is dead tissue and must be digested, and that it is less likely to be tied tightly and strangulate tissues. He feels that with a certain amount of inevitable bacterial contamination infection is more apt to develop after using catgut than silk. Koontz[6] has shown that animal fascia lata preserved in alcohol and washed at operation may be used in exactly the same way as live fascia, thus avoiding the necessity of another operation. He points out on histological grounds that perfect union occurs with the fascia and that the reason that catgut and kangaroo tendon do not act like this is that the collagen fibrils of these

materials are altered both chemically and physically in the process of their preparation. His work, both experimental and clinical, is sound and valuable. Brenizer has made use of threads of live fascia for facial surgery, such as harelip. My experience has been confined to the insertion of living fascial threads to give support and function in facial paralysis. There is certainly a minimum of tissue reaction to this material.

It is obviously desirable to use as little catgut or other material in suturing a wound as possible. By a study of the "Strength of the Healing Wound in Relation to the Holding Strength of the Suture," Howes and Harvey[7] have done a fundamental piece of quantitative work. They have shown that number "0" twenty-day chromic catgut satisfies all requirements of a stitch in fascial and connective tissue layers, while even finer gut than this is sufficient for those structures in which the holding power of the stitch is less.

I am convinced that one should use silk in suturing wounds wherever feasible. It should not be used where there is likelihood of subsequent infection, since it will not be absorbed. In such cases catgut is to be recommended.

Atraumatic Handling of Tissues.—Modern reconstructive surgery has demonstrated that Halsted's emphasis upon extreme care in handling tissues was not overdone. Wounds will always heal more kindly if masses of tissues are not tied off and left strangulated in them, and if fine hooks or non-traumatic tissue forceps are employed throughout.

For the exact type of stitch to be used, I shall refer the reader to any good system of surgery. Claire Straith's modification of the Halsted subcuticular stitch of silver wire in which he uses two running or continuous sutures, one in the dermis and one in the epidermis, has given excellent cosmetic results in the cases in which I have used it. The main points to keep in mind in suturing are to obtain accurate but light approximation of all layers, to control hemorrhage, and to avoid tension on, as well as rolling in, of the skin edges.

FACTORS AFFECTING HEALING IN SUTURED WOUNDS

Certain factors will affect healing in general. Some of these which may be mentioned are age, diet, distant infection and hormones. They have been taken up in this order in a recent article by Anderson.[8] He mentions the work of Clark on diets which shows that a high protein diet shortens the total time required for wound repair. The converse has been proven experimentally by Ravdin and Thompson. In other work which he abstracts, certain vitamins have been shown to affect wound healing quite definitely.

Saitta and Arey both believe that a long-continued vitamin A deficiency delays wound healing. According to Saitta,[9] Anderson, Taffel, and Harvey, treatment with vitamin A, even though only locally, accelerates repair. Horn and Sandor[10] have shown that salves and oils containing vitamin A applied directly to traumatic wounds caused a speeding up of both epithelization and granulation. Lanman and Ingalls[11] have shown that partially scorbutic animals have lowered tensile strength in wounds and that asymptomatic scurvy causes a delay in human wound healing.

Other general measures, such as blood transfusion and nonspecific protein therapy, are of particular significance in the treatment of infected wounds. White and Cooney,[12] under an article entitled "The Nonoperative Treatment of Carbuncles," describe nonspecific protein therapy by injection of milk extract. A milk preparation was used by daily injection intragluteally in 10 c. c. amounts. They summarize their excellent results in 110 carbuncles out of 500, the remainder being treated by other methods. They claim that these injections made operative treatment unnecessary, that the spread of infection is checked, pain is relieved in two to eight hours and healing is hastened.

The favorable influence of periarterial sympathectomy upon the healing of certain types of wounds of the extremities, notably trophic and varicose ulcers, has been pointed out by Leriche.[13]

The first principle to be applied in the postoperative care of a wound is a corollary of the principle of providing contact for the wound edges. This is to put the wounded part (and hence the wound) at rest. If one thinks of any wound as a fracture of the skin just as he would think of the fracture of a bone, better healing will be obtained. Methods to accomplish this vary much. The Logan bow in its employment on a suture harelip is an example of the relief of tension and splinting of a suture line by traction on the surrounding skin. As an apparent exception to the application of the principle of rest comes the opinion of Pack[14] that the heal-

ing of skin grafts applied to excised areas of radiodermatitis will be hastened by active motion of the part. I have had occasion to verify this opinion. Motion should not be instituted until after the graft is firmly adherent and then in moderation.

Considerations of the kind of skin sutures to be employed and the time at which they should be removed should take into account the work of Carrel and Dehelly and later Howes, Sooy and Harvey, which shows that there is a lag period in the healing of fresh wounds of from four to six days. This corresponds to the delay preceding the onset of fibroplasia or fibroplastic reaction which marks the beginning of the true knitting or natural fixation of the wound edges by the growth and union of fibroblasts. During this time the strength of the wound must be artificially reinforced by the use of sutures or by other measures. The latter three workers also demonstrated that the maximum strength of a wound in dogs was attained at the end of ten to fourteen days. I believe that it is safe to say that during the entire time that a wound is attaining its maximum healed strength it should be carefully protected from excessive tension or other injury in every possible way.

POSTOPERATIVE WOUND INFECTION

The treatment of infections arising after the suture of traumatic wounds as well as those after operative wounds will depend specifically upon the nature of the causative organism. One must watch for general and local signs of infection. As soon as fluctuation is determined, it will be necessary to remove accumulated exudate by a proper incision or a separation of the wound edges and to insert a drain. Meleney[15] has pointed out the great advantage of heat in such a condition as this. It may be best applied through warm moist soaks of saline or other mild antiseptics. He feels that this application favors the liquefaction of tissues which have been killed and safely raises the local temperature above the optimum temperature for the growth of organisms. Infection with anaerobic organisms, such as tetanus and gas-forming bacteria, should be treated by antitoxins and proper local oxidizing agents after drainage of pockets. Deep sutures of silk should be removed as far as possible and approximation maintained mainly by strapping with Logan bows as shown in *Fig. 3*. All pockets should be sought and treated by instillation under mild pressure with suitable antiseptics such as Dakin's solution, through Carrel tubes, or by topical application of a chlorine preparation in triacetin or dichloramine in oil. As soon as the infection has been demonstrated by culture to be sufficiently controlled, secondary closure may be practiced as outlined by Carrel and Dehelly.[16] These authors insist upon closure in two stages, waiting for a few days after secondary débridement before the closure of the wound either by suture or by skin graft. This will avoid fresh infection from organisms buried in the cicatrix at the time of its excision. I have found this practice important in dealing with leg ulcers which are to be grafted. Usually a period of four to five days between operations will be sufficient to avoid secondary infection from the cicatrix.

Treatment of Granulating Wounds.—A mere mention of granulation tissue usually brings to the mind of a great many surgeons the idea of antiseptics. The work which I shall report herewith emphasizes the fact that while in certain granulating wounds certain antiseptics may be desirable, in most cases they are highly undesirable and unnecessary. With proper protection against injury, granulations will usually take care of themselves. Reid[17] states that "a protection of granulation tissue from traumatization and insults of any nature becomes of paramount importance in wound healing." In support of this he quotes Billroth's classical experiments on dogs in which he was unable to produce serious infection from daily applying putrid material to a large uninjured granulating surface. The same material injected subcutaneously caused septicemia. In most granulating wounds on the extremities, it is all-important to apply elastic adhesive strapping. I have pointed this out in two previous articles.[18] Granulating ulcers must properly be regarded as fractures of the skin and subcutaneous tissue, exactly comparable to a fracture of a bone. Elastic adhesive provides rest through splinting the skin. The infrequent atraumatic and almost entirely painless dressings accomplished by employing this material are in sharp contrast to the traumatic painful and bloody dressings which until recently were and in certain locations still are the rule when ordinary dressing materials are used.

Antiseptic Treatment.—I do not agree with those surgeons who would belittle the importance of the use of antiseptics in their proper place. Rightly used, they have been of tremendous value in infected wounds. In my opinion they have cut down the time of hospitalization in preparing the patient for plastic procedures.

On the other hand, I realize with Reid that it does not pay to be led away from the tremendous importance in wound healing of "necrosis, débris, devitalized tissue, granulation tissue, fibrin or other factors."

We agree with Newell[19] that, as far as we have seen, the results of giving chemicals intravenously to combat infection in a wound do not seem to be worth the effort. Conceivably this form of therapy may make rapid strides with a proper drug, but at present it seems to be of little benefit. The writer with Drs. Avery and Pilcher[20] has tried the administration of mercurochrome intra-arterially in the limb of a dog in which the circulation was temporarily blocked by tourniquet at the thigh. Quantities were given which would actually stain the skin of the web between the toes, but would not cause necrosis of the limb, in the hope of combating local infection. There was no appreciable evidence of improvement.

As an observer of wounds day by day, I am struck with the great benefit to the patient which may come from an individualization of his particular wound. Close observation in a wound difficult to treat will almost always lead to a rearrangement of local and general measures to the great benefit of the patient. Individualization leads to an honest search for underlying etiological causes, which routine and general antiseptic measures tend to stifle. Perhaps I may best illustrate the result of careful observation of a wound by giving in abstract the record of a case report by Dr. Frank Meleney,[21] which follows:

"*Case IV.*—The fourth case was a private patient in Newark. A year previously she had had an interval appendix removed. The wound became infected and instead of healing it continued to discharge. After six months the infection began to undermine the skin and a large ulcer formed which gradually enlarged by a process of undermining and liquefaction of the margin. It was evident that this case was similar to the other cases." [By which he means to other cases of burrowing abdominal infections which he describes.] "Anaerobic cultivation of the exudate yielded the micro-aerophilic hemolytic streptococcus in pure culture."

It is interesting and inspiring to notice how Dr. Meleney worked at this point. He says: "I now felt certain that this organism was significant and again I prepared vaccine and Besredka filtrate. They were used without any improvement. A wide excision failed to stop its progress. *Fig. 7* [showing a large undermining ulcer of the groin] illustrates the extent of the lesion at that time. Antimony had no effect. Maggots were tried and nearly drove her insane by their activity. The wound improved slightly, but maggots had to be abandoned. Her morale was completely shattered, the pain in the wound could hardly be controlled with morphine, and she contemplated suicide." This description shows that Dr. Meleney was keeping in mind the patient's condition as a whole, rather than her wound alone. Here is the turning point. He says: "In the meantime we had been studying the organism with renewed interest. Its preference for an anaerobic environment suggested the use of a peroxide to inhibit its activity. I asked Dr. Hans Clarke and Dr. Edgar Miller, of the Department of Biochemistry, if they could suggest a peroxide that would yield its oxygen over a relatively long period of time rather than give it off abruptly. After some determination Dr. Clarke suggested zinc peroxide. This chemical has been used commercially for some time as a bleaching powder, but recently the DuPont Chemical Company, of Niagara Falls, has made a fine grade for medicinal purposes which they call 'Z. P. O.' The fact that it would yield oxygen over a period of days and leave the bland zinc oxide with which we are all familiar made the suggestion an appealing one. The idea was carried through at once. A creamy suspension was made in sterile water and applied to the wound. Within three days it was evident that there was a favorable reaction. In a week the whole appearance of the patient and the wound had changed. Her morale had returned, fever and pain subsided, the undermined flaps began to become adherent. Cultures taken from the wound were reported by the hospital laboratory to indicate that the organism was now growing aerobically."

Then follows the description of the further treatment, including the grafting of skin and further treatment of certain deep sinuses with zinc peroxide until healing occurred some months later.

The subsequent careful and brilliant work of Meleney and his coworkers in developing many practical applications for zinc peroxide and in perfecting a technic for its use is highly significant. In his work on this and other cases, we are given a splendid idea of the importance of individualization in leading to the discovery of a specific form of therapy whether it be mechanical, chemical or of some other nature. In this case the remedy depended directly upon the discovery and definition of the nature of the causative agent, namely, the micro-aerophilic hemolytic streptococcus.

Further progress in treating infections must come in this way and not through the efforts of certain commercial houses to push new remedies beyond their specific fields of usefulness. It is unfortunate that human beings should be made the subject of wholesale experiment whenever a drug which has definite merit for certain conditions is placed on the market. The repercussions of a few deaths or tissue necroses from experiments "just because somebody had a 'hunch' to try something out" are a severe and unnecessary setback to progress in the use of antiseptics. I do not feel, however, that such efforts should be allowed to argue against the tre-

mendous importance of antiseptics in combating certain forms of infection.

It is certain that when gangrene of the skin is proven to be due to the action of *Endamoeba histolytica* as in the case which F. L. and H. E. Meleney[22] have demonstrated, recovery will usually follow the administration of a course of "chiniofon" and emetine hydrochloride along with complete surgical excision of the lesion, local application of zinc peroxide, and finally skin grafting.

I am in hearty agreement with the statement of Anderson:[8]

"Many of the reports of common local therapeutic agents are merely enthusiastic clinical impressions."

I might add that reports of wound studies often fail to take into account the favorable influence that the personal interest and attention of the surgeon exert in improving the general care of the patient as well as the local factors concerned in the treatment of his wound.

Only through objective observations, through quantitative studies may we arrive at definite and final conclusions concerning the value of physical and chemical measures to promote wound healing. I wish now to deal with a few of the observations of this nature as applied to wounds. I do not wish to condemn any measures, but I shall not feel that any have proven their worth until quantitative estimates of their effect on wound healing have been made.

I agree with Newell[23] that a careful following of the Carrel-Dakin technic of treating wounds with neutral solution of chlorinated soda (Dakin's solution) offers one of the safest ways of treating massive contaminated wounds. The solution is harmless to the skin if properly titrated and the slight skin irritation which may accompany its use may be prevented by application of vaselined linen strips or zinc oxide ointment. We do, however, agree with Reid[17] that the cells of delicate growing tissues such as the edges of skin grafts may be inhibited in their growth (*Fig. 5*) by overapplication of Dakin's or certain other antiseptics, and that when the wound is found to be covered with a clean fibrin layer or to present clean level granulations, it is best to let it alone until it heals or becomes reinfected, and as he aptly says, "reinfected it will become if it does not heal promptly."

When graulations are healthy, we feel that it is of extreme importance to protect them and as part of this protection to keep them from drying. In a previous article I have pointed out the importance of healing under moist conditions and shown that a semipermeable elastic adhesive is very useful in this connection since it provides a semi-closed system without the usual dead space which so many kinds of dressings unfortunately produce. The importance of elastic adhesive in also providing contact and support in ambulatory cases can hardly be overestimated. In those cases where secretions are abundant at first, I have devised a means as shown in the illustrations of allowing free drainage at the center of the ulcer through almost invisible lengthwise slits; in other words, by fenestration of the strips.

As I see it, the principal advantage of Orr's[24] method of treating osteomyelitis by drainage and subsequent rest in a plaster cast as long as complications do not supervene is that it provides complete rest and protection to the wound, prevents secondary contamination and provides undisturbed moist healing conditions. This may or may not mean a favorable influence on the de-

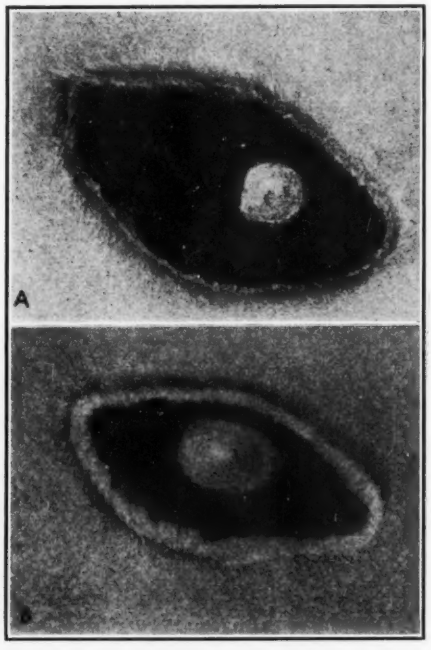

Fig. 5

Retardation of healing by over-dakinization (Reid: *New England Journal of Medicine*, 1936). A. Skin graft on an intensely Dakinized wound. No fibrin. No growth of epithelium. B. Condition of a wound after discontinuing intensive Dakinization. Note coating of fibrin which forms a medium into which new cells will grow. Epithelialization has begun.

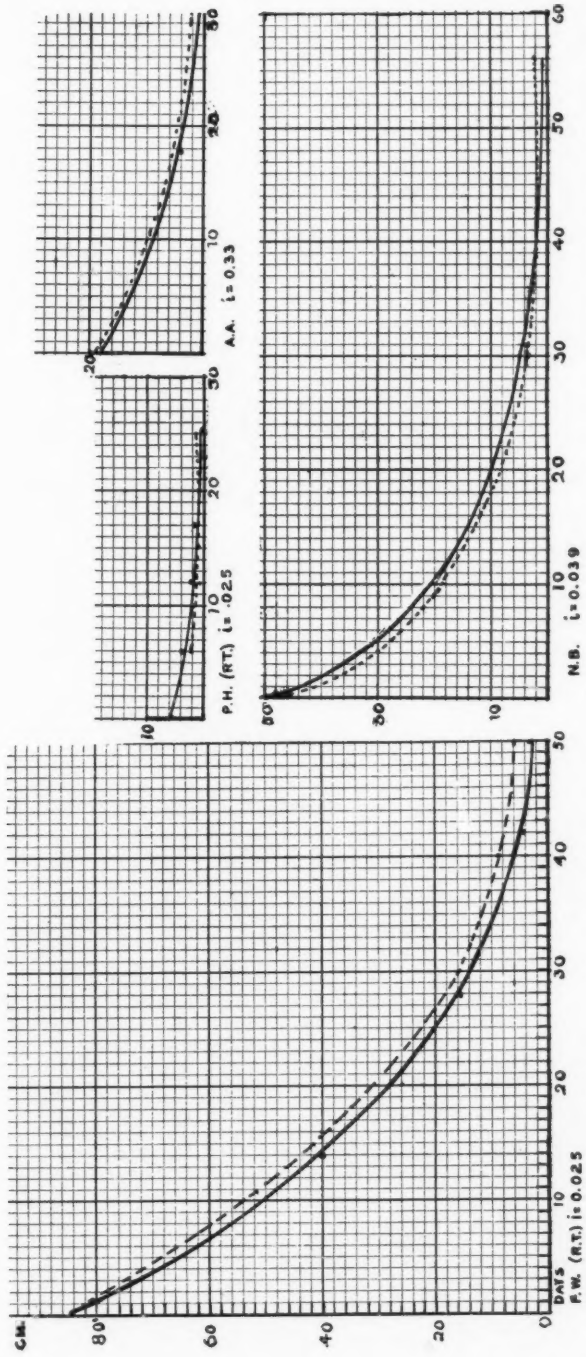

Fig. 6

A critical study of elastic adhesive as a dressing for leg ulcers and other wounds. Solid line = actual curves of healing or cicatrization. Dotted line = ideal curves of healing as computed by Carrel, Hartmann and du Noüy.

velopment of bacteriophage. Until more careful control studies are made, the efficiency of bacteriophage and vaccine therapy must both be held in doubt. In certain cases I feel that both are of value. While not doubting the value of various cod liver oil preparations on granulating wounds in which I have used it and seen it used, I feel that a large part of its beneficial action is exerted through the bland protection it affords and is due to the semipermeable dressing which it provides. Its benefit is partly exerted through its vitamin content according to several authors.

Much study will be required to evaluate the effect of light roentgen rays upon the healing of different types of wounds before definite conclusions may be reached.

Anderson,[8] in his recent quantitative studies on infected wounds treated with various antiseptic agents, concludes that none had any appreciable effect except zinc peroxide, and the latter only in specific cases. On the other hand, he says that no evidence was obtained to indicate that any of the agents in his series retarded healing by excessive chemical irritation on tissue cells.

The quantitative study of wound healing was begun by Ambroise Paré four hundred years ago when he made tracings in order to follow the healing of Monsieur de Vaudeville's leg ulcer. It was continued during the World War when Carrel, Hartman and du Noüy deduced ideal curves of cicatrization for clean surface wounds treated with Dakin's solution.

Employing an elastic bandage spread with Unna's paste on

old chronic ulcers, I have compared their curves with mine. I have thus shown that the rate of healing of most chronic ulcers treated by elastic adhesive is somewhat more rapid than the so-called ideal rate of healing in their wounds. *Fig. 6* is typical of many curves in our series which demonstrate this fact. Thus it is evident that du Noüy's ideal algebraic equal for the rate of healing or cicatrization of infected wounds must be corrected when physical agents rather than chemical antiseptics are employed. I have felt that part of the increased rate of healing which I have proven to exist for ulcers under elastic adhesive is due to the absence of Dakin's solution in my cases, which the originators frankly state is slightly toxic to tissues. Anderson asks the question in this connection:

"Is it not possible that Dakin's solution shortens the healing period of dirty sloughing wounds by hastening the separation of necrotic tissue rather than by any direct antiseptic action?"

This may also be true of the action of carbolic when topically applied to carbuncles. Ray[25] has recently reported excellent results with this drug in carbuncle treatment by injection of small quantities at the layer between the infected columns of fat and the surrounding inflammation area, at a level near the base of the carbuncle.

It would seem that once the sloughing tissue is separated from the wound no antiseptic is desirable, but that healing would be favored by a dressing which afforded protection to the "fracture of the skin" by providing a splinting action at the edges which puts them at rest, which provides further moist healing conditions and which draws the edges inward in such a manner as to favor contraction. I have shown that the strapping of a wound with elastic adhesive accomplishes all these ends provided only that no acute spreading infection is present. Twyman[26] further has shown that "epithelium proliferates readily when subject to a degree of pressure which restrains the growth of granulation tissue. Ferris Smith, employing light inflatable rubber bags over skin grafts, found that the ideal pressure to promote healing was 30 mm. of mercury.

I have mentioned the use of elastic adhesive in granulating wounds, including infected ulcers on extremities, since I feel that with the exception of surgical excision it offers the best chance for cure and the most practical and comfortable dressing.

In very refractive and persistent infection of ulcers, my coworker, Dr. Larsen, has suggested the employment of a 3 per cent ammoniated mercury ointment as a coating for the ulcer followed by its covering with elastoplast locally. This seems to offer much hope. At present he and I are making comparative studies by tracings of wounds under this treatment and those with elastoplast alone. The makers of elastic adhesive have coated the cloth with an emulsion mixed with icthyol for use in certain cases of eczema surrounding ulcers. We have ordered elastic adhesive for trial in which the emulsion is mixed with ammoniated mercury and other substances. However, results have been so good with elastic adhesive strapping alone that we have been reticent to add any drug to it.

In closing I wish to describe a system which I have developed of treating wound surfaces.[27 28] This consists of applying completely transparent air-tight thin rubber and also transparent cellulose-acetate jackets to extremities which present wounds. While affording visibility at all times, these jackets avoid all other dressings to the wound. Pressure within the jacket from the introduction of air will hold skin grafts in contact with the wounded surface, thus doing away with bulky marine sponges, tampons, and so on. This fact has been proven by experimental observations in a test apparatus in the laboratory and is abundantly supported by clinical observations of human skin grafts within the jackets. The physical law on which this is based is embraced in Newton's third law of action:

"To every action there is always opposed an equal reaction; or the mutual actions of two bodies upon each other are always equal, and directed to contrary parts."

The apparatus is in no way to be confused with the apparatus for producing passive vascular exercise. It has been developed particularly for wounds, has a special air-tight seal, and was gradually developed by the author independently over a period of years. By use of these lightweight, inexpensive jackets, the very atmosphere surrounding a wound, the temperature, humidity, solution or gaseous content, positive and negative pressure, and light conditions may be varied at will while the wound is studied through the jacket under direct vision of the naked eye and the total exudate is collected for study. Employing one of these jackets the writer has recently provided a continuous pressure of 30 centimeters of water on four whole thickness skin grafts on the contracted fingers of a child and has followed the take of one graft after another under direct vision. The child's hand was comfortable and the circulation excellent at all times. The four grafts were observed to

be in excellent condition at the end of three days. No other dressings were necessary.

In his poem, "Hudson's Last Voyage," Henry Van Dyke describes Hendrick Hudson as saying to his little son:

"The honor of our life derives from this:
To have a certain aim before us always
Which our will must seek amid the peril of uncertain ways.
Then, though we miss the goal
Our search is crowned
With courage and we find along our path
A rich reward of unexpected things.
Press toward the aim,
Take fortune as it fares!"

The proper treatment of wounds requires that the surgeon shall have a certain aim before him always, namely, to provide conditions favorable to healing. The course may be uncertain at times, but if he will press constantly toward the aim he will discover an unexpected ally in the healing process.

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DISCUSSION (*Abstract*)

Dr. Amos R. Koontz, Baltimore, Md.—I shall not attempt to discuss this paper from the point of view of the plastic surgeon. I am, with every other general surgeon, intensely interested in the healing of wounds, and it is from that point of view that I shall direct my discussion.

There are several points which Dr. Douglas has made which I do not believe can be too strongly stressed. One is the question of too frequent dressings. We frequently see interns dress wounds every day or every other day which would be much better off left alone or dressed every four or five days. Frequent dressings not only do mechanical damage to wounds, but subject them to the danger of outside contamination. When open wounds are dressed, the skin around the wound should be carefully sterilized with a good antiseptic like Scott's solution or tincture of merthiolate. Strong antiseptics in the wound itself do very little good, but if the skin around the wound is kept sterile, continual recontamination of the wound from the skin is avoided. This lesson was learned and amply demonstrated by the French during the World War. I thoroughly agree with Dr. Douglas as to the use of elastic adhesive, and heartily commend him for the part he has played in developing this useful method. The importance of the replacement of small avulsed portions of fingers, ears, and so on, cannot be too strongly stressed. One is continually surprised to find such avulsed portions grow back perfectly in cases in which deformity would result if they were not replaced. The use of strong antiseptics in fresh wounds is not only useless, but actually harmful to the tissues. If the strong antiseptic kills a few bacteria, it also kills many healthy cells besides, and tends to prevent *per primam* healing. A wound that has bled freely and does not contain dirt needs no antiseptic wash whatsoever. If it does contain dirt, this is best washed out with soap and water, followed by ether to remove the soap. Small scratches in children, in my opinion, are best left open. If a strong antiseptic is applied, it causes an exudation of serum, which invites contamination and infection. Such wounds do well, if they are painted with the ordinary aqueous solution of mercurochrome, not for the antiseptic value, but for the metallic protective film that forms when the mercurochrome dries, and thus protects the wound mechanically from contamination.

There was a time when Dr. Douglas' advocacy of silk over catgut would have produced an uproarious protest. Happily, that time has now passed, and the value of silk is being more and more appreciated and its field of usefulness more and more enlarged. There are two great advantages of silk. One is that it can be depended upon to stay where it is put until healing is complete, which is not true of catgut. The other is that there is practically no reaction around a silk suture, the wound being entirely dry, and therefore it is less apt to become infected than the wet wound which develops from the reaction around catgut sutures. It is true that when a wound closed with silk does become infected, some of the silk sutures have to be removed, but with the perfection of modern operating room technic this is no longer the bugaboo that it once was.

The careful handling of tissues is a point that always makes us think of the late Professor W. S. Halsted; and it is one which cannot be too strongly stressed. Tissues that are roughly handled are much more susceptible to infection than those which are carefully handled and left entirely viable. For the same reason when ligatures are applied the smallest possible amount of tissue should be included in the ligature. Dr. Halsted insisted on this, and it was with this point in mind that he invented the Halsted clamp and the mosquito clamp.

Tying off large masses of tissues is bound to produce necrosis, which predisposes to infection.

The period of lag in the healing of wounds is a most important point, and one that is apparently overlooked by many surgeons. Harvey and his coworkers have amply demonstrated that wounds have little tensile strength until ten to fourteen days after they are sutured. I have always thought that this principle is particularly applicable to the after-treatment of hernia wounds. Strong healing in a hernia wound is just as important as the operation itself. For this reason I keep my ordinary inguinal hernias in bed for fully two weeks after operation. I note that many surgeons permit their cases to be up in nine or ten days. It has always seemed to me that this was in violation of what we know about wound healing. I should like to know what Dr. Douglas thinks of this.

The roll of fibrin in wound healing is one that is often neglected by surgeons. Hertzler, of Kansas, showed many years ago (about 1904, I think) that when two intestinal loops are sutured together, fibrin forms in the crease between the two sutured loops, and is responsible for the knitting together of the two peritoneal surfaces. Baitsell, of Yale, in 1916, actually demonstrated in tissue culture the transformation of fibrin into fibroblasts. Many surgeons, as soon as they close a wound, grab a coarse sponge saturated with salt solution and roughly wash the wound off thoroughly. In the light of what we know about the roll of fibrin in wound healing, this practice seems to me to be entirely wrong. If the fibrin is left in place, it not only tends to seal the skin edges mechanically, but plays an important part in the healing of the wound by the production of fibroblasts.

Dr. Fred Y. Cronk, Tulsa, Okla.—We are all vitally interested in the care of wounds. Unnecessary scarring from lack of proper treatment is to be deplored. The time for treatment of wounds is immediately following the accident. I want to emphasize first, as has been very forcibly brought out by Dr. Douglas, the necessity of careful, painstaking cleansing of the wound. By this I mean the removal of all foreign material by gentle wiping and stripping of tissues and especially of the soft tissues where foreign material has been more or less ground in. The use of green soap and water at times applied with a soft brush is very effective. Following this a mild antiseptic may be used further to cleanse the wound. I am very partial to the use of ether not only on the skin margins, but in wiping the deeper tissues as well. It is the careful first procedures that give us primary healing of wounds, and it is this primary healing that leaves the least scarring. Infected wounds and areas where drainage has been used give us very noticeable scarring and often some impairment of function.

It is essential that anatomical structures be replaced in their normal positions, suturing being done with the least amount of foreign material. I have had excellent results with the use of very fine silk and feel that it is superior in many instances to catgut.

Suture of the skin margins should be done with care so that the approximation is most accurate. A little drawing of one margin, or a little increased elevation of one margin over the other, is very noticeable in the healing.

Of the more extensive wounds there is often the necessity for immediate general care of the patient. However, wounds should be immediately protected and an early treatment given, depending upon the general condition of the patient. Early reduction of fractures with proper closure of wounds thereover should be done if we expect the best results. Transfer of tissue to build up deficiencies caused by the loss of substance should be done early.

The physician giving first aid should have a thorough knowledge of tissue repair and if he is not in a position to give the very best treatment at his first visit, he should delay as little as possible in transporting the patient to a place where he himself or a competent surgeon can help the patient most.

Dr. LeRoy Long, Oklahoma City, Okla.—Not only did Paré say "I dressed him and God cured him," but he indicated that he was [illegible] sustained by an inner consciousness, based upon the observation and treatment of many sick persons, that he was able to render good service to the patient. On an occasion when a gentleman expressed great surprise at the recovery of a patient who had been frightfully wounded, Paré said, in substance, to those about him: *"Il a oublié que je marchais dans les salles de l'Hotel Dieu pendant trois ans"*—he has forgotten that I walked the wards of Hotel Dieu for three years.

Just a word about the treatment of lacerated wounds: Some years ago a practical procedure that is of interest to the general surgeon was proposed by Georges Gross, of Nancy, France. He proposed that, in the case of every lacerated wound, a culture specimen be taken and sent to laboratory, followed immediately by the removal of foreign material (épluchage), the cleansing of the wound, and the excision of devitalized tissue. You will observe that the word excision, spelled the same in French and English, is employed. The word débridement has crept into the medical literature of the United States to the extent that it is frequently employed to indicate the excision of tissue. In fact, it is so frequently employed in that sense that one is almost tempted to believe that its wide employment makes it proper. But it is not proper, because, surgically, débridement means the making of incisions to relieve tension, and it is thus employed by every accurate French writer.

When these things are done, that is, the securing of a specimen for culture, the removal of foreign material, the cleansing of the wound and the excision of devitalized tissue, the wound is closed by sutures.

If the laboratory report in the next day or two shows a growth of saprophytes only, the wound is left alone because saprophytes are practically harmless in the presence of sound tissue. If the report shows a growth of staphylococci, it is watched with care, and a suture is removed here and there if there is evidence of beginning infection. If the laboratory reports a growth of streptococci, all sutures are removed and the wound is left open so that there can be free drainage.

Dr. Douglas (closing).—Dr. Koontz question concerning the optimum time for postoperative hernia patients to become ambulatory is most pertinent in the light of the newer knowledge of the increasing strength of tissues as healing progresses. Also very important is the question applied to uncomplicated postoperative appendices and similar conditions. We must not forget that failure to take into account the so-called "lag" or latent period in fibroplasia may predispose to hernia by subjecting injured tissues which are bound together only

by fibrinous adhesions to strains great enough to pull them apart. Dr. Koontz, who has contributed much to the knowledge of hernia cure through approximation of broad surfaces by strong fascial bands, has laid down a splendid rule for his hernia patients which modified might well be applied to all postoperative abdominal cases. He is right in not letting his patients out of bed for fourteen days, for even under ideal conditions, according to Howes, Harvey, Sooy and others, fibroplasia would not be completed in any less time than this. The time saved in the few cases which might "get by" if allowed up earlier could not balance the time loss of recurrence. The soreness which develops as a rule in and about the wound area when patients are let up seven to ten days after abdominal operations is to my mind good evidence that the delicate fibroplastic healing process is being seriously hampered by strain with its accompanying excessive inflammatory reaction. The latter invariably results in a broad weak scar and usually in herniation. One must, of course, admit the probability of a shorter healing time in childhood or under artificial stimuli such as increased temperature just as one must admit its retardation under unfavorable conditions.

Dr. Long's point concerning *débridement* and *épluchage* is well taken. I am afraid, however, that although the latter is the proper term, the former has been used for excision of devitalized tissue so long that it has come to stay.

We are indebted to Dr. Long for stressing the importance of immediate culture. I should like to add that a culture for anaerobes should be included. This will often enable one to get ahead of infections with such micro-organisms in a manner otherwise impossible.

In closing, may I repeat the ideal which I feel should be aimed at in all wound treatment. This is so to increase our knowledge of the factors concerned that our methods will be based upon the fullness of our knowledge as we apply our experience to individual cases.

SURGERY OF SO-CALLED BILATERAL RENAL TUBERCULOSIS*

By Roy B. Henline, M.D., F.A.C.S.
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Mistaken diagnoses of bilateral renal tuberculosis are frequently made, due to technical difficulties in cystoscopy and catheterization of the ureters or to improper interpretation or correlation of the data obtained. As to the treatment of these patients in whom a diagnosis of bilateral renal tuberculosis has been reached, there is a divergence of opinion among urologists, many maintaining that surgery is contraindicated and relegating all these patients to medical treatment, while others believe that some patients may be distinctly benefited by surgery. A careful analysis of the various types of renal tuberculosis would probably reveal that medical treatment alone will benefit some patients, and that either the diagnosis of bilateral renal involvement is incorrect or surgery does benefit some patients even in the face of definite evidence of tuberculous infection of both kidneys.

The types of renal tuberculosis have not been stressed in the scattered clinical reports, nor have any considered statistics available, nor have the results obtained by various forms of treatment been recorded, except in isolated case reports and a few small series of cases. I wish to add a resumé of 40 cases diagnosed as bilateral renal tuberculosis, with particular reference to the difficulties of diagnosis, the treatment employed, and the results after varying periods of time.

It is the consensus of opinion that unilateral, clinical, destructive renal tuberculosis is best treated by nephrectomy, and many statistics have proven the benefit of reasonably prompt, though not hurried surgery. Although such lesions may progress slowly or become temporarily quiescent, no report has been found with pathologic evidence to substantiate the fact that a kidney thus affected will heal under any form of medical treatment.

PATHOGENESIS

Tubercle bacilli first reach the kidneys by way of the blood stream.

The work of Band,[1] Harris,[20] and Medlar and Sessano[37] has demonstrated the preclinical stage of renal tuberculosis. This usually involves both kidneys, with lesions mainly demonstrable in the glomeruli. These lesions may destroy a few glomeruli, but may also heal (Medlar[36]). It has been suggested by Uffreduzzi,[46] Lieberthal,[31] and others that the rich blood supply of the kidney and the large caliber of the renal capillaries cause a peculiar immunity to the tubercle bacilli which may aid in the healing of these microscopic lesions.

These early lesions produce no symptoms and clinically pass unrecognized, although tubercle bacilli may be recovered from the urine at this time. During this stage the pyelograms are normal and the renal function tests show no evidence of renal destruction. A few pus cells will usually be found in the urine (Harris[20]). Since these lesions may heal, leaving only scars in the affected glomeruli, medical treatment only is in-

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*From the Department of Urology (James Buchanan Brady Foundation) of the New York Hospital.

dicated, providing one is fortunate enough to recognize them in the absence of symptoms.

The next step in the development of clinical renal tuberculosis has not been completely proven. However, most observers believe that a small embolic particle of tubercle bacilli reaches the medulla by being washed along the proximal convoluted tubule until a favorable soil for growth of the bacilli is reached, often in the thin arm of the medullary loop (Eberbach;[14]) or that the bacilli reach the medulla by way of the fine efferent glomerular capillaries (arteriolae rectae) which radiate into the medulla. Even these medullary lesions may heal, according to Medlar; but occasionally such a tubercle will spread and finally open into a renal tubule or papilla, discharging its contents into the renal pelvis. When this lesion communicates with the kidney pelvis, the patient has a clinical, destructive tuberculosis, and pathologic evidence of the healing of such a lesion has not been reported.

No explanation has been advanced for clinical lesions occurring in but one kidney when both have been inoculated with tubercle bacilli, yet considerable clinical evidence is available to show that in most instances one kidney alone manifests such lesions. The increasing number of patients who remain clinically well for long periods following nephrectomy indicates the absence of a destructive lesion in the opposite organ. When destructive lesions are found in both kidneys, the discrepancy in the renal damage suggests that the lesions did not occur simultaneously.

CLINICAL LESIONS

The subsequent course of the infection, once the medullary tubercle has ruptured into the kidney pelvis, has been well outlined by Lieberthal.[32] A stricture may occur in the calyx into which the papillary ulcer empties, and stagnation of infected urine in this area results in local renal destruction. Tuberculous involvement of the ureter causes stricture formation, and the remaining calyces become infected and dilated because of inadequate drainage from the renal pelvis to the bladder. The bladder becomes infected and ulcerated and a resulting interstitial cystitis develops which causes contracture of the bladder and diminution of its capacity. The frequency of urination, tenesmus, and strangury produce hypertrophy of the trigonal muscle (Young[50]).

Marion[35] has suggested, and it is our impression, that, as a result of this marked bladder involvement, the integrity of the sphincter of the opposite ureteral orifice is partially lost. During urination a reflux may occur up the healthy ureter, and tubercle bacilli are thus planted in the wall of the ureter or renal pelvis. In this ureter a stricture also occurs and a hydronephrosis develops in the previously healthy kidney (*Fig. 11*). It is our belief that these strictures of the lower ureter are responsible for inability to catheterize either ureter in some patients in whom a diagnosis of bilateral renal tuberculosis has been made. If we assume that marked bladder inflammation and contraction distort the ureteral orifice on the side of the unaffected kidney and prevent the ureterovesical sphincter from functioning normally permitting urinary reflux up the ureter, an explanation would be available to account for the presence of tubercle bacilli in the urine from the uninfected kidney. Due to reflux up this ureter, the ureteral specimen from this kidney may contain urine and bacteria excreted from the diseased organ. Although this occurs only late in some extensive infections, it may well be a cause for incorrect diagnosis of bilateral renal tuberculosis, as suggested by Wildbolz.[49]

DIAGNOSIS

The difficulties in making an accurate diagnosis in bilateral renal tuberculosis have been well recognized by Stevens,[40] Beer,[4] Kretschmer,[27] Marion,[35] and many others.

Finding Tubercle Bacilli.—When one is able to catheterize both ureters, specimens from each kidney may contain tubercle bacilli because of reflux up the normal ureter, and one is misled into assuming that a bilateral renal tuberculosis exists. Conversely, when tubercle bacilli are not found in the urine from the supposedly healthy kidney, by either smear or guinea pig inoculation, one may still be dealing with bilateral tuberculous lesions. Tubercle bacilli appear in the urine intermittently, and failure to find them is inconclusive proof of the absence of a tuberculous infection.

Functional Tests.—Functional tests are helpful in deciding when surgery is indicated, but are of little value in the diagnosis of early bilateral tuberculosis of the kidneys. A small renal lesion may cause no diminution of function demonstrable by our usual functional tests. On the other hand, definitely decreased function may be present, due to a stricture of the ureter and a resulting pyelectasis or hydronephrosis, without tuberculous infection in this kidney. A secondary

pyogenic infection may also supervene and pus cells be found in the urine from this side.

Urographic Findings. — Urograms probably give more information regarding the renal damage than functional tests. Both intravenous and retrograde pyelograms are often necessary. The pyelographic evidence of destructive caseous renal tuberculosis is considered characteristic; yet one may remove such a kidney to find a nonspecific infected hydronephrosis or a renal tumor.[21] When such a definitely destructive renal lesion is shown to exist by pyelogram, and the contour and renal function of its mate are satisfactory, we remove the destroyed organ. These are surgical kidneys, whether or not tubercle bacilli have been found.

TYPES OF INFECTION

Preclinical Infection.—When tubercle bacilli are found in the urine from one or both kidneys, with little or no demonstrable change in the comparative function of each kidney, and no urographic evidence of renal destruction, we treat the patient by medical means only. For, even though we are aware that tuberculosis of the kidney is present, we have evidence that a kidney so affected may heal or at least remain quiescent for many years. Two patients, followed for seven and ten years, respectively, have shown no progression of their disease. Cases of this type are rarely encountered except in sanitaria for tuberculosis, where urine examinations are made routinely on patients with no urinary symptoms. Most of us never see such early stages of the infection because of the complete lack of symptoms. When signs and symptoms develop, there is usually some urographic evidence of a destructive lesion, and surgery is indicated in many instances.

Intrarenal Calcifications.—An occasional patient is seen with bilateral multiple areas of renal calcification. Tubercle bacilli are not found in the urine as a rule, and the comparative functional tests may show equal, yet somewhat diminished, function. The pyelograms will be similar to those seen in bilateral marked pyelonephritis, with long infundibula and dilated and blunted minor calyces (*Figs. 1* and *2*). These calcifications and the lack of evidence of destructive caseous lesions probably represent nature's attempt to heal the infection. The urographic findings usually permit one to make a diagnosis of bilateral renal tuberculosis, and these patients frequently go for years under medical care with only slight progression of the disease. Surgery is not indicated.

Bilateral Clinical Infections.—One should be guarded in telling a patient with bilateral renal tuberculosis that his infection is bilateral and

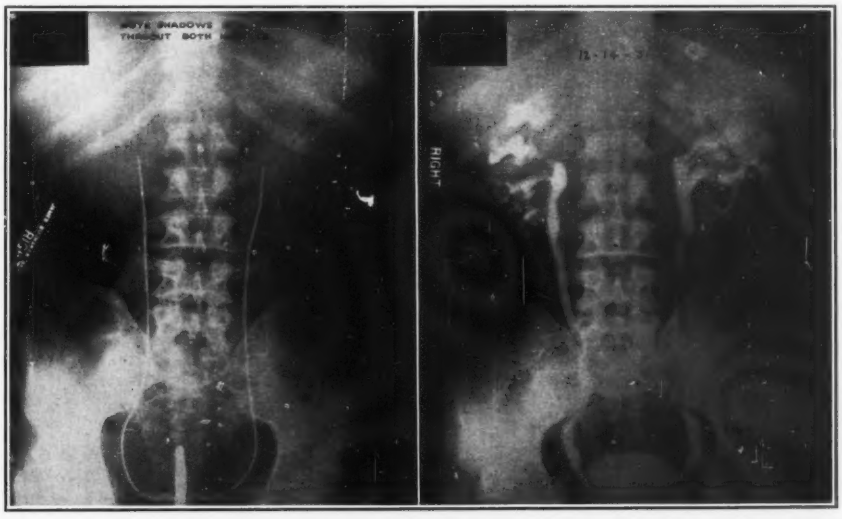

Fig. 1

Bilateral renal tuberculosis. Note the calcified areas scattered equally throughout both kidney areas in December, 1931. Tubercle bacilli were recovered from each kidney. With similar lesions in each kidney, medical treatment only is indicated. Nature apparently has attempted to heal this lesion.

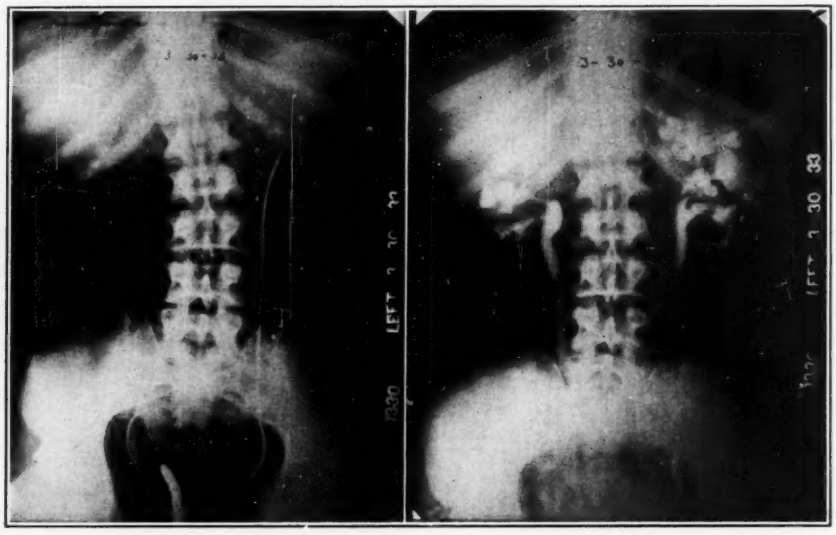

Fig. 2
Same as Fig. 1, in 1933. Note larger calcifications and evidence of further renal damage.

that surgery is not indicated. There is a small group of cases in which this opinion may later have to be reversed. We have seen 5 patients, who were considered inoperable when first seen, yet, after a period of adequate medical care, presented evidence of a pyonephrotic kidney on one side and a regression of the infection on the other, and were considered operable. To convince these patients that they should then be operated upon was difficult.

Searching for Tubercle Bacilli.—Many urologists search repeatedly for tubercle bacilli from the apparently normal kidney before removing the other, definitely proved tuberculous kidney which has been shown to be partially or completely destroyed. Thomas and Kinsella,[42 43 44] Begg,[5] Hale,[18] Caulk,[10] Barney and Jones,[2] Herman and Greene,[22] with many others, believe that one should be as nearly certain as possible that the other kidney is not infected with tuberculosis before one is justified in removing the badly infected organ. To this end, they often perform repeated cystoscopies to collect urine from the normal kidney, for staining and guinea pig inoculation, before removing the diseased kidney.

Emmett and Braasch[15] believe that repeated cystoscopic examinations to search for tubercle bacilli from either kidney are rarely necessary, and should not be done in most cases. The finding of a definitely destroyed tuberculous kidney on one side and a functionally efficient mate is evidence enough, they claim, for removal of the destroyed kidney even though some tubercle bacilli may be found in the urine from the other kidney. Marion,[35] Kretschmer,[27] and many others concur in this opinion. Wildbolz[49] rarely searches repeatedly for tubercle bacilli from the "normal" kidney, but says that bacilli may be found in the urine from the "normal" organ because of reflux from the bladder, tuberculous nephritis, or the fibrotic form of tuberculosis, and relies on functional tests for differentiation of these types from caseocavernous disease.

Urograms.—The urograms in most cases of bilateral renal tuberculosis show, on one side, a dilated, irregular renal pelvis and ureter with shaggy, moth-eaten minor calyces in the kidney, and a very small lesion or none at all in the other kidney pelvis. The appearance of the pyelogram depends partly on the stage of progression of the disease in each kidney. In some instances one kidney may be functionless, as seen from the intravenous urogram, while the pelvis of its mate may show varying stages of dilatation. Occasionally, when neither ureter can be catheterized, one must rely on an intravenous urogram alone, and, fortunately, much suggestive information can be obtained in this way, as has been pointed out by Braasch.[7] Whenever possible, we per-

form bilateral retrograde pyelography, and have noted no ill effects from this procedure. Questionable distortions and small areas of infection are more clearly delineated on retrograde pyelograms.

FREQUENCY OF BILATERAL LESIONS

Although one would expect clinical bilateral tuberculosis to be commonly demonstrated, because of the pathogenesis of the infection, it is relatively infrequent. Bumpus and Thompson[9] reported 43 positive guinea pig inoculations from supposedly normal kidneys in 175 patients suffering from destructive renal tuberculosis in the opposite kidney. Braasch and Scholl[6] reported 7 similar results in a series of 22 cases. Legueu[30] collected 87 cases of bilateral lesions from the literature and added 6 of his own. He concluded, from his results, that removal of the poorer functioning kidney was justified by the resultant relieving of the distressing symptoms and the prolonging of life. Israel[25] found the second kidney infected with tuberculosis in 1.6 per cent of 1,022 patients who had had an early removal of a kidney for tuberculosis; whereas 29.2 per cent of those who had not had an early nephrectomy subsequently had bilateral involvement. Barrie[3] found bilateral renal tuberculosis in 14.6 per cent of patients clinically, but at postmortem the incidence of bilateral infections was increased to 47.9 per cent. Caulk[10] found bilateral involvement in 31 per cent of 263 cases of renal tuberculosis, and Barney and Jones[2] in 24 per cent of their cases. In 500 autopsies reported by Greenberger,[17] 45.6 per cent of the subjects showed miliary renal tubercles, but only 4.8 per cent clinical caseous lesions.

There is being reported an increasing number of cases that have been diagnosed as bilateral renal tuberculosis, in which the patients have either been much improved or clinically cured. Cases of this nature have recently been reported by Bugbee,[8] 2; Morse and Braasch,[38] 19; Begg,[5] 1; Cirio,[11] 6; Decker,[12] 3; LeFur and Laminaud,[29] 3; Jacobs,[26] 4; Gile,[16] 8; Donati,[13] 3; Viannay,[47] 3; Reynard,[39] 2; Marini,[34] 10; Strachstein,[41] 1; Lazarus,[28] 2; Thevenot,[45] 19. This makes a total of 86 patients suffering from bilateral renal tuberculosis who have had the destroyed kidney removed, 58 of whom are reported to be much improved and some clinically cured.

Caulk[10] believes that the reported good results are mistaken diagnoses, but the mortality of the reported cases of bilateral renal tuberculosis that did not have the benefit of surgery is very high. Of 84 patients with bilateral lesions, reported on by Bumpus and Thompson,[9] 49 were dead in a short time without surgery. Morse and Braasch,[38] reporting the comparative results following surgery in 19 bilateral and 90 unilateral infections, show much improvement or cure in 11, or 64.7 per cent, of the bilateral cases and similar improvement in 45, or 64.28 per cent, of the unilateral infections. He concludes that the mortality and improvement following surgery is about the same in unilateral and bilateral renal tuberculosis. Begg[5] concludes that it is better to remove an obviously tuberculous kidney than to search too long for conclusive evidence of infection in the other.

REPORT OF CASES

I wish to report, and summarize, 40 cases diagnosed as bilateral renal tuberculosis, realizing that the diagnosis may be questioned in some instances, due to the technical difficulties encountered. However, a correlation of all the available clinical evidence with the course of the infection and autopsy study of patients who, in life, had manifested similar clinical signs,

Fig. 3

Same as Figs. 1 and 2, in 1938. Calcified areas are much larger and several calcium phosphate calculi have been passed to the bladder. His general condition has remained good under medical care, although he complains of urinary frequency and intermittent colic. No tubercle bacilli are demonstrable in the urine at present.

makes the diagnosis of bilateral renal tuberculosis seem likely in these cases.

Age and Sex.—The average age of these patients was 34.4 years, the oldest being 63 and the youngest 17 years. There was approximately an equal number of males and females in this series.

Family and Past History.—Only 5 patients gave a positive history of tuberculosis in their immediate families, and only 15 were aware of any previous tuberculous infection in themselves (*Chart 1*).

Chart 1

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

	Age Years	Sex	Family History	Personal History
Average	34.4	M. 21	Positive 5	Positive 15
Oldest	63	F. 19	Negative 33	Negative 22
Youngest	17		Not stated 2	Not stated 3

Symptoms.—Most of these patients had had symptoms longer than 6 months and had been treated for cystitis. One patient had had mild urinary symptoms and tubercle bacilli in his urine for 35 years before he came under our observation (*Chart 3*).

Chart 2

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

SYMPTOMS

Frequency	— 33	Urgency	— 1
Hematuria	— 12	Tumor	— 1
Nocturia	— 8	Pain in bladder	— 1
Pain in kidney	— 7	Pyuria	— 1
Burning	— 7	Tenesmus	— 1
Dysuria	— 3	Retention	— 1

All of the patients sought medical advice because of bladder symptoms except 2, who complained primarily of renal pain (*Chart 2*). Frequency was complained of by 33 patients, hematuria by 12, nocturia by 8, and burning on urination by 7. More than one symptom was usually present. There is no characteristic symptom of

Chart 3

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

DURATION OF SYMPTOMS BEFORE TREATMENT

Less than 6 months	3
Six months to 1 year	12
One to 3 years	13
More than 3 years	12
Longest duration	35 yrs.

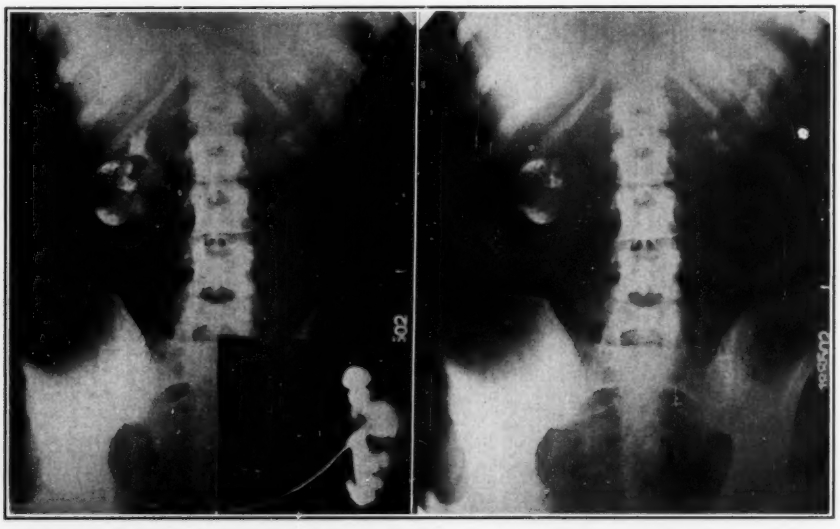

Fig. 4

Bilateral renal tuberculosis with calcification. Tubercle bacilli found in bladder urine. Left kidney, with little remaining function, removed. There have been no tubercle bacilli in bladder urine since operation. The bladder and general condition are much improved since operation one year ago.

bilateral renal tuberculosis, the foregoing complaints being the most common symptoms in all forms of urinary tuberculosis.

Diagnosis.—The accuracy of the diagnosis in some of these cases may well be questioned, since we were unable to catheterize either ureter in 14 cases and one ureter in 7 others (*Chart 4*).

Chart 4

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

CATHETERIZATION OF URETERS

Could catheterize both ureters	19
Could catheterize only one ureter	7
Could catheterize neither ureter	14

The diagnosis was therefore based on the cystoscopic appearance of the bladder, the finding of tubercle bacilli in the bladder before and after operation, the presence of tuberculosis in the removed kidney, urographic evidence of tuberculosis in each instance, and the course of the disease since the institution of treatment (*Chart 6*).

Stained smears of urine from the bladder showed the presence of tubercle bacilli in 34 of the 40 patients before operation (*Chart 5*). After nephrectomy or medical treatment we were still able to find tubercle bacilli in the catheterized bladder urine of 20 patients at one or more times (*Chart 8*). These patients frequently go for months without demonstrable tubercle bacilli in their urine. Organisms may be found on one occasion and be absent at the next examination. Only 4 patients had demonstrable tubercle bacilli in their urine at their most recent examination. Three of these had had one kidney removed, 10, 4 and 2 years ago, respectively. The fourth patient has been considered a nonsurgical case, and has been under medical treatment for

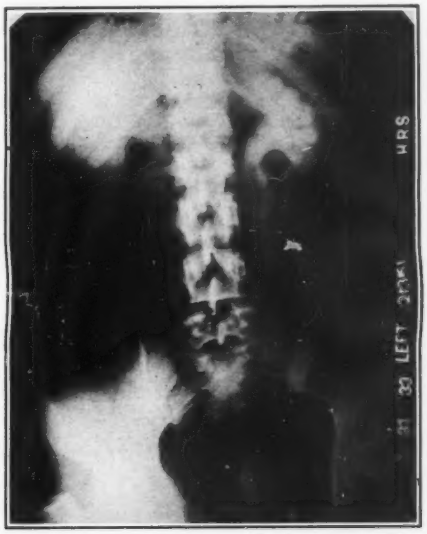

Fig. 5

Bilateral renal tuberculosis with functionless right kidney; age 29. At 3 years of age he had tuberculosis of the left hip and pleurisy at age 17. Note small contracted bladder which made cystoscopy and ureteral catheterization impossible. Right tuberculous kidney removed in February, 1933. No tubercle bacilli have been found in the bladder urine since operation. He has received medical care and his general health is good, but he still complains of frequent urination, five and a half years after operation.

Chart 5

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

FINDINGS AT BEGINNING OF TREATMENT

	Bladder		Right Ureter		Left Ureter		Diagnosis Made Without Finding Tubercle Bacilli
	Tbc. Stain	Guinea Pig	Tbc. Stain	G. P.	Tbc. Stain	G. P.	
Operated Cases 29	Positive 27 Not found 2	Positive 2 Not done 27	Positive 1 Negative 1 Not done 27	Positive 2 Not done 27	Positive 1 Negative 1 Not done 27	Positive 1 Not done 28	1 had tbc. kidney removed
Nonoperated Cases 11	Positive 7 Not found 4	Positive 2 Not done 9	Positive 2 Not done 9	Not done 11	Positive 2 Not done 9	Not done 11	3 treated medically
Per cent	Positive 85	Positive 10					

5 years, with a history of symptoms for 10 years previously (*Chart 8*).

Guinea pigs inoculated with bladder urine gave positive evidence of tuberculosis in 2 patients in whom we were unable to find the organisms in the smear. One of these patients had had a tuberculous kidney removed, and died 3 years later from tuberculosis of the remaining organ. The other patient was not considered to be surgical, and has been under medical treatment for 5 years, reporting only slight vesical discomfort and excellent general health (*Chart 6*).

Thus, in 36 of the patients tubercle bacilli were demonstrated in the urine from the bladder. We have been unable to find tubercle bacilli in the 4

Chart 6

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

DIAGNOSIS MADE BY

(1) Tubercle bacillus in bladder urine by smear or guinea pig		36
(2) Urograms considered characteristic of tuberculosis		
(3) Appearance of bladder suggestive or diagnostic		
(4) Kidneys removed showed tuberculosis		29
(5) Eight deaths:		
2 generalized tuberculosis	Autopsy	
1 tuberculous meningitis	Autopsy	
1 renal tuberculosis	Autopsy	
2 generalized tuberculosis	Autopsy not done	
1 uremia, hydronephrosis	Autopsy not done	
1 cerebral hemorrhage, no operation	No autopsy	

Chart 7

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

DIFFICULTIES IN DIAGNOSIS

(1) Bladder inflammation, diminished capacity	
(2) Unable to catheterize either ureter	14
(3) Unable to catheterize 1 ureter	7
(4) Finding tubercle bacillus in both kidney urines strongly suggests bilateral lesion	
(5) Absence of tubercle bacillus in both kidney urines cannot eliminate tuberculosis	
(6) Must rely on urograms. Is hydronephrosis due to stricture or renal tuberculosis?	
(7) Surgical specimen or autopsy	
(8) General course of patient's condition	

Fig. 6

Bilateral renal tuberculosis. Note functionless left kidney and multiple calcified lymph nodes. Urine from right kidney showed pus 4+ and tubercle bacilli. Left nephrectomy in November, 1934. Bladder urine still shows tubercle bacilli, but he is working and his general health is reported good, four years after operation. He has a tuberculous urethral stricture.

Chart 8

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

CONDITION AFTER TREATMENT

	Bladder Urine More than 1 Month Postoperative		Living Average Time Since Treatment Began	Dead Average Time Since Examination or Operation
Operative Cases 29	Positive	17	21 patients	7 patients
	Negative	9	5.4 years	5.15 years
	No specimen	3	Lost sight of 1	
Nonoperated Cases 11	Positive	3	8 patients	1 patient
	Negative	3	7.56 years	2½ years
	No specimen	5	Lost sight of 2	
Latest Urine Smears Showed	Positive	4	3 surgical, 10, 4, 2 years after operation 1 medical, 5 years after Rx	
	Negative	28		

remaining patients. Two could not be traced, 1 died from cerebral hemorrhage 5 months after he was first seen, and the fourth had a tuberculous kidney removed 5 years ago and now complains of slight frequency of urination, although he is working regularly and his general condition is good (*Chart 5*).

We have been compelled to rely on evidence from the pyelogram or intravenous urogram, which we considered to be characteristic of tuberculosis, for the diagnosis in some of these patients in whom ureteral catheterization could not be carried out (*Chart 6*). However, the 29 kidneys removed at operation were all tuberculous.

Treatment and Results.—Twenty-nine patients had surgical removal of one kidney. Seven of these, after living an average of 5.15 years, have died (*Chart 8*). Two succumbed of generalized tuberculosis 4 and 9 years, respectively, after nephrectomy, 1 of tuberculous meningitis five weeks after operation, and 1 from tuberculosis of the remaining kidney 3 years after operation. Autopsies were not obtained in the other 3 cases. Two, however, were diagnosed as generalized tuberculosis before death, 3 and 4 years, respectively, after removal of one kidney. The third patient was reported to have died from uremia secondary to a hydronephrotic kidney 12 years after removal of the tuberculous organ (*Chart 6*).

Twenty-one patients have survived an average of 5.4 years since removal of the more diseased kidney. Of these, 10 are reported to show marked bladder improvement or to be practically cured of symptoms (*Chart 11*). In 13 the general condition has been improved since operation.

Eleven patients have been considered nonsurgical, either because both kidneys were equally infected or because their renal lesions showed some calcification and a fibrotic form of tuberculosis. Two have refused operation. Of these 11 patients, 8 have lived an average of 7.56 years, 2 have been lost sight of, and 1 died of cerebral hemorrhage 2½ years after treatment was begun. Of the 8 nonsurgical patients who are still living, 4 have shown marked bladder improvement and 5 are in better general health (*Charts 8* and *11*).

It is interesting to note that in this series there were 5 patients, considered inoperable when first examined, who, after medical care, were later advised to have one kidney removed (*Chart 9*). In these cases, after periods ranging from 6 to 24 months (averaging 16 months), one kidney became markedly destroyed and the other showed evidence of improvement. It was believed that the general condition, as well as the resistance, of these patients would be improved by removing the badly infected kidney. Three were operated upon, and died from tuberculosis in an average of 3.4 years. Their general condition and bladder symptoms were temporarily improved after operation, but it is difficult to judge whether their lives were prolonged or shortened by surgery. If surgery was indicated in these 3 patients, and we believe it was, one must explain these deaths by the amount of destruction in the remaining kidney, the virulence of the infection, or the lack of resistance of the patient. The other 2 patients refused operation and have lived an average of 5.5 years under medical treatment. They both complain of some frequency of urination and their urine contains tubercle bacilli, but they are working and their general health is reported to be good.

It would appear from these statistics that medical treatment offers the longest life expectancy

Chart 9

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

INOPERABLE, BECAME OPERABLE

Patients	Date Symptoms Began	Date Medical Rx Started	Date Considered Operable	Date Kidney Removed	Time Since Operation	Present Condition
(1)	1927	4/1933	12/1934	12/1934	3 years	Died general tuberculosis
(2)	1929	10/1929	10/1931	10/1931	4 years	Died general tuberculosis
(3)	1927	3/1928	9/1928	9/1928	3 years	Died renal tuberculosis
(4)	1929	6/1930	2/1932	Refused	6.5 years	Improved, urine positive
(5)	1923	5/1933	3/1934	Refused	4.5 years	Improved, urine positive

Patients operated upon lived average of 3.4 years.
Patients not operated upon still living average 5:5 years.

and the lowest mortality. However, one must consider that the renal lesions treated surgically are usually not comparable to those that have had medical treatment alone. The surgical cases are the destructive, infected, caseous lesions which frequently result in a functionless kidney and, if not removed, merely remain as a focus of infection.

This improvement under proper medical care, in cases of bilateral renal tuberculosis considered to be nonsurgical, has been due primarily to the splendid work of Dr. Wang,[48] who is in charge of our clinic for the care of inoperable and post-operative cases of genito-urinary tuberculosis. His conscientious and untiring efforts in the handling of these difficult and often hopeless cases has been manifestly rewarded by the results he has obtained in some of these patients. As just mentioned, however, the patients treated medically are those without marked caseation or pyonephrosis. When calcification and fibrosis are present in the kidney, there usually is only slight infection or little pus in the urine. Nature has already attempted to heal the infection and the patient's resistance is good. These patients are not as ill from toxic absorption as those whose kidney lesions have been broken down, often with some secondary infection and inadequate drainage to the bladder. Most of the surgical patients have also had the benefit of postoperative medical care.

Only 15 of these 40 patients, when first seen, had demonstrable tuberculous lesions elsewhere or gave a history of any previous signs of tuberculosis. Several had more than one tuberculous focus. Epididymitis had developed in 6 patients when first observed, and was found to be present eventually in 14 of the 21 male patients (*Chart 10*).

DISCUSSION

The clinical tuberculosis of the urinary tract which we see differs in some respects from that observed in a sanitarium for tuberculosis. Only about 30 per cent of our patients give a history or show evidence of any pre-existing tuberculosis, while patients in a sanitarium are there primarily for treatment of a tuberculous infection elsewhere in the body. In our present series 37.5

Chart 10

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

ASSOCIATED LESIONS

		Tuberculous		Non-tuberculous	
Right nephrectomy	14	Epididymitis	14	Mastoid	1
		Prostate	3	Thyroid disease	1
		Tbc. joint	6	Bladder stone	1
Left nephrectomy	15	Pulmonary	7	Ureteral transplant	1
		Pleurisy?	2		

Chart 11

ANALYSIS OF 40 CASES DIAGNOSED BILATERAL RENAL TUBERCULOSIS

RESULTS OF TREATMENT

		Bladder Symptoms					General Condition			
	Traced, No.	No Change	Slight Improvement	Marked Bladder Improvement	Practically Cured		Improved	Same	Worse	Dead
Cases operated upon	29	28	3	8	6	4	13	7	1	7
					47.6%		61.9%			
Cases not operated upon	9	9	2	2	2	2	5	2	1	1
					50%		62.5%			

Fig. 7

Bilateral renal tuberculosis with ankylosis of right hip. Cystoscopy impossible, but bladder urine contained tubercle bacilli in 1933. He became critically ill in 1935 and refused surgery. During this period of severe pain in the left kidney region and high fever, he suddenly voided more than 250 c. c. of pure pus from his bladder and his temperature soon became normal. His left kidney apparently had drained down the ureter. Urine still contains tubercle bacilli with frequent urination, but his general health is good, and he is working.

Fig. 8
Bilateral renal tuberculosis. Removal of right kidney four years ago with improvement (Case 2).

per cent gave evidence of other foci of tuberculous involvement. Although the tubercle bacilli reach the kidney from some focus of infection, by the time we see the patient the primary focus has usually healed so completely that it cannot be detected by our customary physical and x-ray examination. Most of the patients who come to us with tuberculosis of the urinary tract are robust, healthy looking individuals, with no evidence of a generalized infection, either active or latent. Although we are aware of the possibility of extension of the infection elsewhere in the body, our patients, when first seen, usually present a lesion localized in the genito-urinary tract. If they have suffered from a tuberculous lesion elsewhere, which has healed, their resistance may be greater than that of sanitarium patients, who may have other active lesions and must resist more than one focus of infection.

The preclinical, *asymptomatic*, bilateral lesions which heal are rarely seen outside a sanitarium for tuberculosis, and most of them would probably not be diagnosed except in a sanitarium, where patients are suffering from other tuberculous lesions. When patients come to us, they have not only passed this stage, but have often shown an ability to heal the original focus of infection. A patient with symptomatic genito-urinary tuberculosis is a potentially surgical case, and should be subjected to the careful scrutiny of a competent urologist and an internist trained in the treatment of genito-urinary tuberculosis. This type of the disease, with symptoms and no evidence of healing, should not be confused with the early, asymptomatic lesion which does heal. The treatment of these two types of lesions is different and has been confused or misinterpreted in the recent literature.

The history, in most cases of bilateral renal tuberculosis, from a clinical standpoint begins with a unilateral destructive lesion. There have been many instances of unilateral renal lesions, that have not been operated upon, in which later there has been involvement of the other kidney. Since our only hope of preventing this complication is to remove the destructive lesion, most urologists agree that this should be done. Although this procedure rarely, if ever, is an emergency operation, in the absence of definite contraindications it has been our practice to remove this infectious focus as early as possible. To follow the progression of such a lesion for a period of time, or to delay its removal while repeated cystoscopic examinations, looking for tubercle bacilli from the opposite kidney, are being made, seems to us unjustified. Some of these lesions

Fig. 9
Single tuberculous kidney eighteen years after removal of right kidney for tuberculosis. Bladder contracted to 30 c. c. capacity. Note dilatation of lower calyx and ureter. This x-ray taken before transplantation to sigmoid (Case 3).

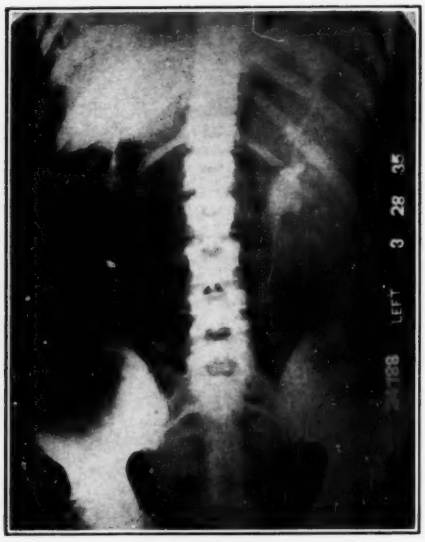

Fig. 10

Same as Fig. 9, three and a half months after transplantation of ureter into sigmoid. Note pelvis and ureter are diminished in size (Case 3).

may remain quiescent for prolonged periods of time, especially under ideal medical care, only to become active at a later date. Distressing bladder symptoms may develop or progress while one is watching such a lesion. Removal of the focus of infection may permit the patient better to control his other lesions, if any are present, or may prevent dissemination of the toxins or tubercle bacilli from this area. Toxic nephritis and nephrosis have been reported to develop in the opposite kidney from such a lesion. It has been suggested that tubercle bacilli may be carried in the blood stream to the opposite kidney.[49] Marked bladder involvement may permit a reflux up the healthy ureter, which, in turn, becomes infected. In order that these sequelae may be prevented if possible, the infected kidney should be removed within a reasonably short time after the diagnosis has been made.

Hunt,[24] Beer,[4] and MacKenzie[33] are among those who believe that the worst kidney in bilateral renal tuberculosis should not be removed except for pain, hematuria, or sepsis, providing the other is only slightly involved. We agree with this point of view, but believe that sepsis of varying degrees is very frequently present from an infected, destroyed kidney. This sepsis or toxicity may not be manifested by fever, general malaise, or other definite subjective symptoms, but more often adversely affects the other kidney or aggravates the bladder symptoms.

In the small series of cases reviewed here, there is some evidence of improvement in patients believed to be suffering from bilateral renal tuberculosis in whom the badly infected kidney was removed. We are fully aware of the high mortality and morbidity in these cases, but since other methods of treatment have proven of little benefit, surgery has been offered them. Gile[16] believes that one should remove the more damaged kidney, since this offers the only chance of relief and may possibly result in a cure. Kretschmer[27] says that there have been times when he has had no choice but to operate under these circumstances. Several of our patients have been made more comfortable and may have had their lives prolonged by the timely nephrectomy followed by proper medical care.

Whether the least infected kidney develops a hydronephrosis from a tuberculosis in the kidney itself, or from a tuberculous ureteral stricture caused by reflux of tubercle bacilli up the ureter, is difficult to state. From the frequency with which we were unable to catheterize either ureter, although the better kidney showed only moderate damage, it might be assumed that a

Fig. 11

Bilateral renal tuberculosis with functionless left kidney which was removed ten months previously. Note stricture of lower right ureter with hydronephrosis and hydro-ureter (Case 4).

low ureteral stricture occasionally occurs as the earliest demonstrable lesion on the less infected side. It may be that a destructive tuberculous lesion of the better kidney occurs only late in the disease.

So-called auto-nephrectomy occurred in 10 of these patients. Eight were operated upon, 2 dying 3 years later; the remaining 6, operated upon from 1 month to 6½ years ago, have shown some improvement. Two patients have not been operated upon and have improved under medical care for 5 and 35 years, respectively. All of the removed kidneys showed that the tuberculous process was still active even though in some instances the ureter was completely occluded. Since these kidneys also are potential foci of infection, which may become active, we advise their removal. Beer[4] has cited many instances of patients with exclusion of one kidney (auto-nephrectomy) who later have returned for treatment of infection in the other kidney.

Medical care is a valuable adjunct in the treatment of these patients, yet we remove a kidney as soon as the diagnosis of a surgical lesion has been demonstrated. Medical therapy is more efficacious after the infectious focus has been removed. To attempt to build up a patient by sending him to the country or a sanitarium, before the infected area has been removed, has not been found advisable in those whose lesions are considered to be surgical. Only when the bilateral lesions are of nearly equal severity, as proven by functional tests (when possible) and urograms, or when the function of the less infected kidney is markedly diminished or insufficient to carry on life, is medical treatment carried out immediately after the diagnosis has been established.

Dilatations of the remaining ureter, following nephrectomy, have been done in 7 patients. Tuberculous strictures in these ureters are frequently seen; and in 2 patients pain in the remaining kidney has been relieved by dilatation of the ureter, and in 3 infection has been decreased. Some patients are distinctly benefited by thus affording drainage from these secondarily infected kidneys. Hunner[23] has reported beneficial results in similar cases.

Bladder instillations of gomenol have been used with some success. Filling the contracted bladder with a very dilute solution of bichloride of mercury to the point of pain has resulted in increasing the bladder capacity and relieving the distressing vesical symptoms in some instances. Twenty-seven of these patients have had bladder dilatations by some method. Drs. Wang and Lowsley have devised a quartz lamp which may be inserted into the bladder, so that ultraviolet rays may come in direct contact with the vesical lesions. The bladder is emptied of fluid and distended with air during this short exposure. Several patients have insisted that they were definitely benefited by this therapy. In this series 30 patients have received quartz light therapy, either intravesically or to the skin or both. Old tuberculin has been given to 26 patients. A special dietary regimen has been followed by 30 patients. Dr. Wang, who is in charge of this work, follows all these patients with a sympathetic understanding which they need.

In the past the condition of patients with bilateral renal tuberculosis was considered to be hopeless. For many years proved unilateral tuberculous kidneys have been treated by nephrectomy followed by medical treatment with definite beneficial results. After summarizing the 40 cases in this series we now feel that bilateral renal tuberculosis is far from hopeless and that some of these patients may be definitely benefited by both surgical and medical treatment under the combined guidance of a urologist and an

Fig. 12

Ankylosed hips and bilateral renal tuberculosis. Right kidney functionless and partially calcified (autonephrectomy) and small lesion in upper calyx of left kidney (Case 5).

internist. Although we do not know whether their lives have been actually prolonged by any form of treatment, there is evidence that many of these patients, who formerly were considered hopeless, are made more comfortable by combined surgical and medical treatment.

SUMMARY

(1) A resumé of 40 cases, diagnosed as bilateral renal tuberculosis, is presented.

(2) The difficulties in making an accurate diagnosis have been stressed. Cystoscopy is often difficult or impossible, due to marked bladder involvement. Ureteral catheterization was impossible in 14, or 35 per cent, of the patients. One ureter could not be catheterized in 7. Finding tubercle bacilli in the urine specimens from each kidney may not be conclusive proof of bilateral involvement. The absence of tubercle bacilli in the urine from each kidney does not eliminate the possibility of tuberculosis.

(3) Tubercle bacilli were found in the bladder urine, by smear of guinea pig inoculation, in 36 of the cases.

(4) To make a diagnosis, urography must be relied on, assisted occasionally by the finding of tubercle bacilli in the bladder urine.

(5) Twenty-nine kidneys removed at operation and autopsy of 4 patients all revealed tuberculosis.

(6) Twenty-one of 29 patients have lived an average of 5.4 years following removal of the more infected kidney in so-called bilateral renal tuberculosis. Seven have died, after living an average of 5.15 years, while 1 patient could not be followed.

(7) Eight of 11 patients have lived an average of 7.56 years with medical treatment alone. One died 2½ years after treatment was begun, and 2 were lost sight of.

(8) Patients who were treated by medical means alone manifested a clinically different type of renal tuberculosis (fibrotic) as a rule. A few destructive lesions thus treated (rare) have shown no demonstrable progression.

(9) Ten, or 47.6 per cent, of the living patients, who were operated upon, showed marked bladder improvement or were practically cured after removal of the worse kidney, while 13, or 61.9 per cent, have shown general improvement. Four, or 50 per cent, of those treated medically alone showed bladder improvement, and 5, or 62.9 per cent, improvement in general health.

(10) One patient, with a diagnosis of tuberculosis in her remaining kidney, had her ureter transplanted to the sigmoid 4 years ago, with complete relief of intolerable vesical pain.

(11) Prompt removal of an obviously destroyed kidney is justified provided the function of its mate is sufficient, as this is the method of treatment offering the greatest hope of relief.

(12) Repeated search for tubercle bacilli from a functionally and pyelographically normal kidney, in the presence of a destructive tuberculous lesion in its mate, has not been done.

(13) Tuberculosis of the urinary tract was a local disease in 25 of our 40 patients, with no evidence of active lesions elsewhere in the body.

(14) So-called auto-nephrectomies may be closed off from the bladder, but the renal lesion usually remains pathologically active, and, being a focus of infection, should be removed.

(15) The hydronephrosis and hydro-ureter which frequently develop in the better kidney may be the result of primary tuberculosis of the kidney or secondary to a ureteral stricture.

(16) Dilatations of the ureter of the remaining kidney may relieve renal pain or decrease the signs of infection by affording better drainage.

(17) Medical care by an experienced internist is indispensable in the postoperative care of bilateral renal tuberculosis, as well as in the treatment of patients suffering from bilateral inoperable tuberculosis.

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THE CONSERVATIVE TREATMENT OF CONGENITAL CLUB FEET IN INFANTS*

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The popularity and security of modern surgical methods have led to their widespread adoption by busy surgeons, replacing in some instances the slower and more conservative forms of treatment. We believe that in the treatment of congenital club feet, over emphasis has been placed on forcible manipulations under anesthesia or open operation, and that certainly in the treatment of club feet in infants these methods have no place. The purpose of this paper, therefore, is to reiterate the plea for conservative treatment of congenital club feet by presenting a summary of the cases treated in the club foot clinic during the past one and one-half years.

Inspired by the work of Dr. J. H. Kite,[1] and believing in the principles clearly outlined by him, we have adopted at the Crippled Children's Hospital the entirely conservative method of treating congenital club feet in infants. Although we believe this method to be applicable to older children, this paper will be confined to children in the infant age group, that is, one year or less. In the clinic we have had, as far as possible, all the club foot cases report for treatment on the same day, feeling that this not only facilitates the work of the surgeon, but also has a psychological effect upon the parents because they can see other children than their own under treatment.

When a child is first seen in the clinic, an x-ray and photograph of the feet are made as a record and a careful inquiry as to whether there are any other congenital deformities in the child's parents or near relatives made. The parents are then told of the method to be used and it is explained that the treatment will extend over many months, and that faithful cooperation is necessary on their part for successful use of this method. The conscientious surgeon will realize the responsibility he assumes in treating a con-

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Fig. 1

Anterior-posterior x-ray of untreated club feet showing the os calcis and talus directly superimposed. At this time only three tarsal ossification centers are showing, the os calcis and talus posteriorly and the cuboid anteriorly.

genital club foot, and it will be his duty not only to follow his case through until it is completely corrected, but also to instruct the parents to the point where they understand the problem and are cognizant of their obligation to the child's welfare.

Correction of the deformity and restoration of normal or near-normal alignment has frequently been the aim of surgeons treating congenital club feet, but we feel that this is only a half-result for it should include restoration and maintenance of function to be regarded as a good and complete result. It is not denied that anatomical restoration can be obtained by forcible manipulations under anesthesia, or by operative surgery, but it can be said that these methods often result in stiff, inelastic, painful feet; feet that may be anatomically good but functionally poor. Here in early childhood are being developed the two foundations upon which the patient must walk for the rest of his life and this prospect obligates the surgeon to obtain for his patient feet that will function. Being convinced, therefore, that the older methods were not giving us good feet, we adopted the conservative method and have felt amply repaid by the end results. These show not only restoration of form but preservation of function as well.

Anatomy.—In the infant foot, whether deformed or not, all the elements are complete, but the bones are only partially ossified and are rather an orderly arrangement of cartilaginous masses. Of the seven tarsal bones, only the calcaneus, astragalus, cuboid and external cuneiform show centers of ossification; the others not making their appearance until two or three years later. This very malleability of the infant foot makes it at once a semiplastic medium which can be transformed by gentle pressure. These facts underly the slow conservative correction of congenital club feet, a small amount of correction being obtained each time the cast is changed. Gentleness is the keynote, for too much force exerted here will result in fracture of the cartilaginous surfaces or rupture of tiny ligaments, either of which may give rise to hemorrhage and ultimate interosseous fibrous adhesions. Such adhesions impair motion and make the foot much less serviceable than it might otherwise be.

Technic.—The method of applying and wedging the casts will be gone into in some detail for in their correct application lies the comfort of the patient and the successful guidance of the correcting force. We have used only short leg casts, feeling that they are more easily applied, more economical, and just as efficient as long casts. We have never had a short leg cast slip

Fig. 2

Anterior-posterior x-ray of club feet after the adduction and inversion have been corrected, showing the os calcis and talus to be separated at their anterior ends. When above position has been obtained, the equinus may then be corrected by bringing the foot straight up in dorsiflexion. The ossification center for the external cuneiform has now appeared on the medial side of the cuboid.

Fig. 3

Lateral x-ray showing a "rocker-bottom" deformity, obtained when the foot is brought into dorsiflexion before the adduction and inversion have been completely corrected. Note that although the anterior foot is flat, the posterior foot is still in equinus; and that the normally straight plantar surface has been broken in the midtarsal region.

off when an adhesive agent is used on the leg under the padding. Some authors insist that long leg casts should be applied to correct inward rotation or torsion of the tibia, which torsion frequently accompanies club foot deformity. We feel that any corrective force in plaster aimed at correcting torsion of the tibia is lost in the knee joint. This tibial deformity may best be corrected by a rotation osteotomy after the feet are completely corrected.

The foot is first gently manipulated and the point of correction for that visit determined so that after the cast is applied the amount of correction held in the plaster will be just a little short of that obtained by manipulation. Attention to this point will prevent any areas of pressure necrosis. The foot and leg are next painted with a light coat of either shellac or compound tincture of benzoin, the latter being less irritating, although both substances are quite satisfactory as adhesive agents and much less expensive than liquid adhesive. One layer of cotton flannel is next wrapped from the tips of the toes to the knee, each winding being carefully and smoothly applied while the foot is held in the corrected position. With all the toes flat and in close approximation to each other, the plaster is started around them and the forefoot is finished at this point, thus allowing the surgeon to hold the correction while the rest of the cast is being applied. Ordinarily it requires one-half roll for the forefoot and one and one-half more to finish the cast, each roll being either two inches or three inches wide, depending upon the size of the baby, and between six feet and seven feet long.

The adduction (varus) and inversion are corrected first, no attention being given to the equinus until these first two elements of the deformity have been corrected. There are several reasons for this procedure and this order must be adhered to if good results are to be obtained. Inversion and eversion take place in the subastragalar joint and as the inversion deformity is to be corrected first, the foot must be left in equinus, the position in which the subastragalar space is open and most relaxed. In addition, the navicular (scaphoid) must be brought around from the medial side of the head of the talus (astragalus) to its normal position directly in front of it so that the weight thrust will be in a straight line from the talus through the navicu-

Fig. 4

Case of severe congenital equinovarus treated entirely by the conservative method. Note the preservation of the longitudinal arch in the corrected feet.

lar to the toe. If this part of the deformity (adduction) is not completely corrected, the deformity will recur when weight is permitted because the thrust will fall obliquely on the navicular and the foot will be driven around into adduction again. It has been our experience that adduction is the hardest component to correct. It is usually the chief deformity, recurring in the feet which relapse. If the inversion of the os calcis is not corrected, and the foot brought up into dorsiflexion, the still inverted os calcis will ride up against the medial side of the talus and further pressure will result not only in a tight binding of these two bones together, but also a final break in the midtarsal joints with a resulting "rocker bottom" foot.

Value of X-rays.—Dr. Kite, in making the primary analysis of the above factors, has demonstrated how the x-ray may be used in determining when the inversion and adduction have been corrected and when the foot is ready for the final correction in dorsiflexion. An x-ray picture is made with the knees straight up and the feet flat, with the tube being pointed straight down on the feet. If the os calcis and talus are separated by a small space at their anterior ends, the foot is ready for dorsiflexion. If their shadows are still superimposed (as they are in the original club foot position), the feet should be replaced in abduction and eversion until the correct position is obtained.

When the foot is ready for dorsiflexion, it is brought back directly in a mid-position between the malleoli and a cast applied, molding well up under the os calcis and holding the toes flat. Next a wedge of plaster is removed anteriorly, the cut being made at the level of the malleoli, the lower edge being parallel to the sole of the foot and the upper edge coming upon the tibia. When this gap is gently closed and held by a figure-of-eight plaster, the heel cord will be stretched and the foot will be protected by a plaster slipper formed by the lower part of the cast. In our earlier cases we had a high percentage of relapses because we did not check the feet by x-ray before completing treatment, and depended on club foot shoes to hold corrected feet which should have been held in plaster. We now not only check all feet routinely with x-ray, as outlined above, but hold them longer in plaster, even after full correction has been obtained, and then put the children in straight last shoes. It is felt that club foot shoes cannot and will not correct deformities, nor prevent their recurrence, and if the deformities have been completely corrected they are not necessary.

The following figures are a summary of 70 cases treated in our club foot clinic:

(1) Males predominate over females, 70 (49 cases) to 30 per cent (21 cases).

(2) Bilateral involvement is more common than unilateral, bilateral 60 per cent (42 cases), unilateral 40 per cent (28 cases).

(3) Types of deformity

	Per cent
Equino-varus	87
Calcaneo-valgus	8
Varus only	2
Mixed	2

(4) Average age at beginning of treatment was 4.4 months.

(5) Average length of time under primary treatment was six months, the casts being changed completely every two weeks.

(6) Positive history of other congenital deformities in same family was found in 11 per cent of our cases.

(7) Rate of recurrence has been 11 per cent to date.

CONCLUSIONS

(1) We believe that a method of treatment that uses a series of plaster casts and wedgings without the use of an anesthetic, and without forcible manipulations and operations, is of great value.

(2) Our experience is recorded as additional evidence in favor of this method.

(3) No originality is claimed, either for the method or the principles outlined above, but it is hoped that the advantages of the conservative method will be appreciated and widely adopted by surgeons treating club feet.

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THE SURGICAL MANAGEMENT OF DIVERTICULITIS*

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While Cruveilhier[1] first described the formation of diverticula in the colon ninety years ago, it was not until after the beginning of this century that sufficient clinical experience was accumulated to make possible any real knowledge of the condition. Before 1900 only scattered reports were available; after this period observations recorded from the surgical field were added slowly; just how slowly is indicated by the fact that W. J. Mayo,[2] in 1907, reported a total of only five cases which had been operated upon for this condition at the Mayo Clinic. Since 1914 our information has been materially increased by the development of better diagnostic facilities.

While there is no reason to presume that the actual incidence of diverticulosis and subsequent diverticulitis has increased except in so far as all diseases common in older individuals have increased because of the present longer life span, the number of proven cases is now much greater each year. The better understanding of the use of the sigmoidoscope and the more frequent application of the double contrast colon x-ray are unquestionably responsible for the larger number of individuals in whom the lesions are now demonstrated. Moreover, there has come about a considerable improvement in the management of the condition, including a differentiation of those in whom medical treatment is satisfactory from those in whom surgery is imperative. It is now possible to differentiate malignancy from the obstructive lesions produced by diverticulitis, and it is therefore no longer pardonable to subject patients with peridiverticular tumefaction to hazardous one-stage resection of the portion of the colon involved, on the presumption that the lesion might be malignant or might be a combination of diverticulitis and malignancy.

It is difficult to estimate the incidence of diverticulosis in the general population, although some autopsy figures have indicated that as many as 5 per cent of individuals over 40 present this phenomenon. It is known that from 3 to 10 per cent of all those who have gastro-intestinal x-ray studies are found to have diverticulosis of the colon. Hayden[3] reports that in 6,426 patients on whom routine barium enemas were done at the Massachusetts General Hospital, 3 per cent were found to have the disease; the highest figure is that given by Spriggs and Marxer,[4] of London, who reported that 10 per cent of 1,000 cases presented some evidence of the condition when complete gastro-intestinal studies were made. Russell Haden,[5] of Cleveland, tells me that these figures are closely approximated at the Cleveland Clinic. It is believed that approximately one-fifth of individuals with diverticulosis ultimately progress to the stage of diverticulitis. While the mechanism by which these herniations develop in the wall of the bowel is not understood, certainly the condition is a disease of the latter decades of life. In a series of forty cases which I have recently analyzed, 73 per cent were over 55 years of age. The condition is more common in males than in females and it is still apparent that a sedentary life and a tendency to obesity tend to exaggerate the symptoms.

When Louis Wilson,[6] in 1911, found four cases of carcinoma in association with diverticula of the colon in fifteen specimens, he was convinced that this association was more intimate than had been previously thought. The publication of his work and other similar observations were no doubt responsible for a general opinion which obtained for some years that carcinoma was likely to be found in association with inflammation of diverticula. The study of larger series of cases in more recent years has indicated that this relationship is so rare that it may be regarded as purely coincidental. Since carcinoma and diverticulitis are both lesions which occur chiefly in the left colon and in elderly individuals, it is to be expected that both lesions would occasionally occur simultaneously. Rankin and Brown,[7] who found only four accompanying carcinomas in a series of 227 cases of diverticulitis, believe that this parallelism of lesions has been over-emphasized in the past, and there is no doubt that it has been responsible for much ill-advised surgery.

It is still believed that diverticulosis is a condition which presents no symptoms and that it is

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only when an inflammatory reaction occurs that clinical manifestations are produced. Willard and Bockus,[8] however, described a syndrome which they found associated with diverticulosis and which they interpreted as being due to this condition. This syndrome included intermittent diarrhea, constipation, epigastric pain or distress, flatulence and general abdominal pain. Two explanations may be offered for the findings of these observers. The first is that the picture described is that which is also seen in the unstable colon and secondly that it is difficult, if not impossible, to rule out the presence of a mild degree of inflammation in such diverticula as may be present.

The characteristic symptoms of diverticulitis when once definitely established are pain, low grade fever, slight leukocytosis, flatulence and slight nausea. The pain is usually in the left lower quadrant of the abdomen and may be relieved by expelling gas. The removal of an innocent appendix during this stage is a not unusual nor unpardonable occurrence.

It is my opinion that the passage of blood from the bowel has been under-emphasized as one of the symptoms of diverticulitis. The prevalent opinion concerning the incidence of bleeding as a direct result of diverticulitis is well expressed by Rankin and Graham[9] in their recent text:

> "Blood in the stool is of little significance in diverticulitis, and although it is present in a number of cases, usually proctoscopic examination will reveal that the presence of the diverticulitis has little, if anything, to do with the presence of the blood."

Hayden,[3] in his recent book, is equally emphatic that

> "Bleeding should not be considered a symptom of either acute or chronic diverticulitis. Although it was present in 28 of our 140 cases [at the Massachusetts General Hospital], there is no proof that the blood actually came from the involved area."

On the other hand, Brown and Marcley,[10] while stating that they had reason to believe that such instances of bleeding as occurred in individuals with diverticulitis under their observation were due to anorectal disease, were willing to admit that it was possible that they had been overcritical in their interpretation of the cause of bleeding, and they called attention to the fact that both Ochsner and Bargen[11] and Willard and Bockus[8] have reported that bleeding occurred in 7 per cent of their cases.

Bleeding has been a frequent concomitant or presenting symptom in individuals with diverticulitis coming under our observation. I have examined twenty-four consecutive case records obtained from the recent files of Baylor Hospital to determine the incidence of this particular symptom in this group. These patients were seen by various members of the medical or surgical staff. Bleeding had occurred in six cases; in two of these there was an apparent possibility that other lesions were responsible, leaving an incidence of 16 per cent. The patients in this series were sufficiently ill to be in a hospital and the incidence of bleeding could be expected to be higher than in those seen routinely in the office of a gastro-enterologist. While they were not all examined proctoscopically, the presence of diverticula and the absence of higher causes of intestinal bleeding were established by careful roentgenograms. In a series of sixteen cases seen by us since 1935 we have, however, complied with every diagnostic necessity to eliminate any other cause for bleeding from the bowel when it occurred. Each of our patients has been sigmoidoscoped several times, all have been checked by roentgen ray and have had the benefit of medical consultation. If bleeding is a symptom of diverticulitis, this group of sixteen cases should be expected to show a higher percentage of incidence of hemorrhage from the bowel, as they were referred to us with a question of surgical intervention or because of unexplained bleeding. Eight of the sixteen patients gave a history of hemorrhage which was not explained by any other lesion. In four of the patients the blood was slight in amount and either mixed with the stool or with mucus. In four others it was spontaneous, massive and dark. The incidence of blood in the stool in the forty cases analyzed was 30 per cent. We have seen no cases which required surgery because of hemorrhage alone, and here our experience checks with that of Hayes,[12] who has reported two massive hemorrhages from diverticulitis which cleared up under medical management. While there should be no relaxation of the attempt to determine the presence of some additional lesion which might produce bleeding, particularly cancer of the bowel, I feel that the prevalent opinion that diverticulitis never bleeds is thoroughly unwarranted.

The surgical management of diverticulitis is concerned chiefly with the detection of the presence of its various complications and their correction. Mild inflammation of diverticula is usually satisfactorily controlled by medical meas-

ures, including diet, heat, and hot normal saline irrigations of the left colon, preferably given with the patient in the knee-chest position. It is only when the lesion has progressed to the stage of peridiverticulitis with beginning obstruction, perforation, abscess formation, adhesion or fistula to the bladder or adjacent viscera that the intestinal surgeon should properly be called in.

The diagnosis of diverticulitis with a complication is one which cannot be made from a textbook nor from the use of any one of the diagnostic procedures available and each problem presented is one for individual study and judgment. Certainly the clinical history and the physical examination may not be relied upon entirely. A long-standing diverticulosis without symptom may suddenly blossom into a complex pathological situation or a mass discovered in the lower left abdomen may be one of several lesions. However, the combination of a low grade temperature, tenderness in the left lower quadrant, sigmoidoscopic evidence that the lower colon is somewhat fixed, especially in an individual over 50 years of age, are sufficiently suggestive that careful radiographic study should be made on the colon, preferably by the contrast technic.

I have seen the statement that sigmoidoscopy is of little value, but within recent years the reverse of this has been found to be true. While in no way does it approach the usefulness of the x-ray as a diagnostic method, it does have a definite place and should be carried out, preferably before, and in addition to studying the colon roentgenologically. The procedure should be relied upon for the additional information that can be gained by it rather than looked upon as a competitive method inferior to x-ray. It may be expected to show immobilization on sacculation of the left colon, the tiny mouth of a diverticulum, presence or absence of anorectal disease, the presence of an inflammatory exudate or blood coming from a higher area, or it may reveal a malignant tumor. As I have already pointed out, the presence of blood in the sigmoid, while valuable to eliminate a lesion in the rectum or anal canal as the source of the bleeding, does not rule out the possibility that the condition is a diverticulitis.

Accurate diagnosis can be confirmed only by the barium enema. The advantage of the administration of the opaque medium in this manner rather than by mouth is that it prevents additional intestinal stasis above the lesion and that the information may be acquired much more quickly. Uncomplicated diverticula are filled with the barium and appear on the film as rounded outpouchings in relief along the border of the bowel. Diverticulitis is manifested by varying degrees of spasm and hypermotility of the segment involved, which usually is the sigmoid (*Fig. 1*), together with varying degrees of obstruction due to the narrowing of the lumen of the bowel by the fibroplastic reaction or by the spasm which may be sufficiently marked greatly to reduce the caliber of the lumen. In contrast to carcinoma, the segment of bowel involved by diverticulitis is usually longer and, unlike the sharp line of demarcation frequently showing some degree of umbilication between cancer and normal bowel, the limits of the lesion do not terminate abruptly but assume, as they merge into the normal lumen of the bowel, a fusiform or spindle shape character (*Fig. 2*).

It has been believed that the presence in an x-ray film of one or more diverticula tends to establish the lesion as being benign rather than malignant. Unfortunately this is not true. A certain number of individuals with carcinoma

Fig. 1
Peridiverticulitis of sigmoid with obstruction, showing spasm of bowel near lesion and extent of narrowing of lumen.

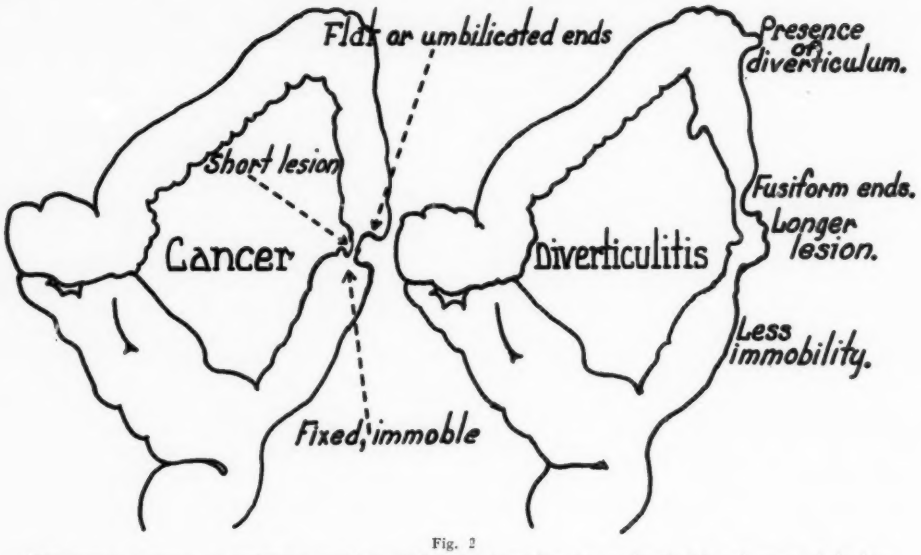

Fig. 2
Diagrammatic summary of contrasting radiologic findings in obstructions due to diverticulitis and cancer of the large bowel.

of the colon have diverticulosis in addition. At my request, Mr. Cuthbert Dukes,[13] pathologist to St. Mark's Hospital in London, was kind enough during my visit in his institution to review the specimens in a recent series of one hundred cases of carcinoma of the left colon and rectum which had been removed by a sufficiently extensive procedure (the perineabdominal resection) that a large amount of adjacent bowel was available for study. He found that in 12 per cent of this series of carcinomas the bowel also was the seat of a diverticulosis or diverticulitis, not, of course, in direct association with the malignancy. It is therefore quite important that the differential diagnosis shall not be made on this evidence alone.

Present day opinion regarding the indications for surgical interference is more conservative than at least some earlier writers expressed. We now limit surgical maneuvers to the complications arising from a long-standing chronic peridiverticulitis and cases of an acute type in which local catastrophes have occurred or are impending. Moreover, even here the tendency is toward conservatism and we now believe that a temporary colostomy alone should be the first step in surgical treatment unless abscess formation requires drainage in addition. If obstruction is sufficiently complete, preliminary cecostomy, to be followed in a few days by colostomy, may be the procedure of choice. Almost never may the descending colon be used for colostomy nor should any but the gentlest exploration on the left side be attempted for fear of inducing peritonitis. Moreover, if resection of the involved area is indicated later, a sufficient length of bowel is not available for both procedures unless the intestinal vent is placed in the transverse colon. In the past, colostomies done on the transverse colon have presented certain mechanical inconveniences to the patient and to the surgeon. The liquid character of the fecal current in this portion of the colon is difficult to control and excoriates the skin when the usual double-barrelled colostomy is constructed. Moreover, effective closure by means of the Mikulicz clamp is much more difficult here than on the left side. For that reason we now believe that the technic for doing a colostomy in the transverse colon described by Sir Hugh Devine and modified by Alton Ochsner presents many advantages over the usual type heretofore performed. By the Devine colostomy the left side of the colon is completely defunctioned so that no fecal material may enter the distal stoma; liquid feces are not constantly discharged on to the skin, causing

erosion and irritation, and the closure may be carried out with only minor additional surgery. The procedure as originally described by Devine[14] consists of making an upper right rectus incision, suturing together the adjacent surfaces of a loop of transverse colon to form a long spur, passing clamps through stab wounds, one on either side of the longitudinal incision, to grasp the bowel which is then severed between the clamps with a cautery, after which the severed ends are drawn through the two stab wounds. The outstanding disadvantage of this procedure was the danger of contamination of the wound and trauma of the blood supply of the colon at the time the severed ends of the bowel are drawn through the stab wounds. To obviate this, Ochsner and his associates[15] have modified the technic by using a transverse incision down to the fascia so that the two stoma may be brought out at either end of the wound and the skin sutured between.

If the length of the transverse colon does not permit this procedure, the hepatic flexure may be mobilized and the ascending colon used for the proximal loop. The clamp may be removed from the proximal bowel within 24 to 48 hours, but the clamp is left on the distal loop for about one week, which retains the peritoneal surface of the bowel in close approximation to the abdominal wall, permitting firm adhesion.

When healing has progressed sufficiently, the lower opening permits copious warm saline or antiseptic through-and-through irrigations of the affected bowel. The colostomy should be permitted to function until any subsequent operative procedure is completed, or if no further surgery is needed, because of resolution of the process, the colostomy should not be closed until the lumen has become practically normal, which requires a period of from nine to twelve months. When closure is indicated, it is done with a special clamp which crushes out the long spur, followed by a simple local inversion of the ends of the bowel.

It is emphasized that only the gentlest exploration is permissible at the time the colostomy is done. If an abscess is present, it should be drained; if the mass is adjacent to the bladder, it is sometimes wise to place the omentum between the two structures to avoid fistula formation.

A surprisingly large percentage of individuals with obstruction or even small sinuses will recover completely from colostomy alone. If obstruction continues, or if the possibility of malignancy still persists, resection may be done after a period of at least six weeks. If a fistula exists into the bladder and fails to heal, resection of the diverticular mass, together with suprapubic cystotomy and suture of the bladder opening, may be necessary.

In the past the prognosis for diverticulitis with complications has not been highly favorable. Radical surgical procedures without preliminary colostomy, injudicious exploration and manipulation, failure to recognize and drain pericolonic abscess were no doubt responsible for many of the operative deaths.

The responsibility which rests upon the surgeon has been emphasized by the fact that preliminary diagnosis is now possible in a great majority of cases. This diagnosis may not be made by a radiologist alone nor by a clinician alone, but the combination of their findings carefully considered should result in sufficiently accurate conclusions to permit the planning of a safe method of intervention which should materially reduce the mortality.

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VARIATION IN MORPHOLOGIC REACTION TO INJURY*

A DISCUSSION OF CERTAIN ASPECTS OF THE PROCESS OF INFLAMMATION, WITH SPECIAL REFERENCE TO THE LARGE MONONUCLEAR WANDERING CELLS AND FACTORS GOVERNING THEIR RESPONSE TO INJURY.

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For many reasons which I shall not attempt to enumerate and for the soundness of which I shall certainly hold no brief the pathologist has come to be looked upon by many of his clinical colleagues as somewhat of a magician. Among his paraphernalia he is believed to carry a bag of tricks which his magic wand, his microscope, may produce at will for complete elucidation of the most complex problems. Being one of those supposed magicians and, therefore, reasonably familiar with the inner working of pathological "magic" and furthermore being ultra conscious of the fact that the microscope is an instrument which magnifies problems about as often as it solves them, I have felt that I might interest you by exposing, as it were, one of the pathologist's tricks. While what I shall have to say about variations in morphologic reaction to injury may disillusion you with regard to one phase of the pathologist's black magic, you will recall that the magician who wishes to continue his trade never gives away his best tricks, not even to his fellow tricksters.

Progress toward rational therapy, the ultimate aim of all of us, requires at least a working conception of the nature of disease. While one may make many definitions of disease, the simple biological expression *reaction to injury* tells about all that is fundamental to any definition. For the pathologist, physical, chemical and morphologic changes in response to tissue stimulation constitute disease. This holds likewise for the clinician, although his efforts in the role of diagnostician and therapist are largely devoted to study of the outward expressions of reaction to injury.

In order to make diagnosis and therapy as easy and workable as possible, our medical forbears made many practical rules of thumb from simple clinical observations and by applying such rules came to recognize what we call disease entities. The end result of this struggle to simplify and to be what is called practical is that our thoughts about disease are now expressed largely in terms of disease entities rather than in the more fundamental terms of biological reaction to injury. By the selection of the subject, "Variation in Morphologic Reaction to Injury," for this presentation I thought that I might renew your interest in the somewhat abstract subject of the nature of disease. Although in so doing I am conscious of diverting many of you from your more usual interests, I hope to be able to show you that the matter is quite practical as well as of intriguing scientific interest.

It is impossible to deal here with more than one of the essential elements in our conception of the nature of disease. I have chosen to place emphasis upon reaction rather than injury for the reason that no disease is possible, regardless of the extent or degree of injury, if the tissues injured are incapable of responding to that injury. I would further emphasize reaction since we know now that the capacity of injured tissues to respond is distinctly limited while the number of agents capable of producing injury is virtually unlimited. If this be recognized, then the various disease entities with all their individual variations may be considered to arise either through simple variations in detail of each of the fundamental reactions to injury or out of the many combinations in which these reactions may appear. If, therefore, one would attain an understanding of any of the common disease entities, a familiarity with the possible variation in reaction of living tissues is inseparable.

Just what, one may ask, constitutes this all-important reaction of tissues? The answer may be put quite simply. When injury occurs, one of three things takes place. The tissue either submits, wherein it usually succumbs; or it resists through an aggressive or progressive process; or it alters itself in such a way that its function continues in a modified form. In other words it adapts itself to the new conditions under which it must live. While reaction in itself implies function and while function is involved in a profound way in each of the above type responses, I shall limit my remarks to a discussion of morphologic reaction only and particularly to that which one sees in inflammation, a response to injury which I prefer to think of as a mechanism

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of active resistance. Even in this day of emphasis upon serums, vaccines, and specific chemical therapeutic agents I dare say that the body possesses in the inflammatory process a protective mechanism that makes a veritable pygmy of any artificial therapeutic method. The enormous importance of the inflammatory reaction is attested by the continuous appearance of contributions dealing with the subject and by the frequent, almost heated, debates which periodically arise over the meaning and significance of features of this reaction which were well known even to Hippocrates.

The effectiveness of an inflammatory reaction as a defense mechanism rests specifically upon its intimate structural composition. In almost any inflammation one may find the following essential morphologic elements, all of which are derived from the host (*Plate I, Fig. 1,* and *Plate II, Fig. 5* and *Fig. 6*).

(1) Wandering cells, that is, cells that do not in the ordinary sense form a part of the permanent or fixed structure of the involved tissue.

(2) Fluids, in excess over the normal tissue content.

While one may be interested by the gross character of the inflammatory process which is determined in general by the quantitative variations in these essential elements, the point of greatest interest and significance in inflammation is the fact that the appearance and proportion of the essential elements are dependent upon specific factors at least some of which are now very well known. Adequate treatment of variations in morphological reaction is impossible without discussion of each of the above-mentioned elements. Since, however, time does not permit this, I must be content to deal with the single factor of the wandering cells and that very briefly.

The wandering cells arrive at the injured area either by way of the circulating blood and lymph, or they wander into the affected area from nearby tissues through the ameboid activity appropriate to them, or lastly they arise *in situ* through a

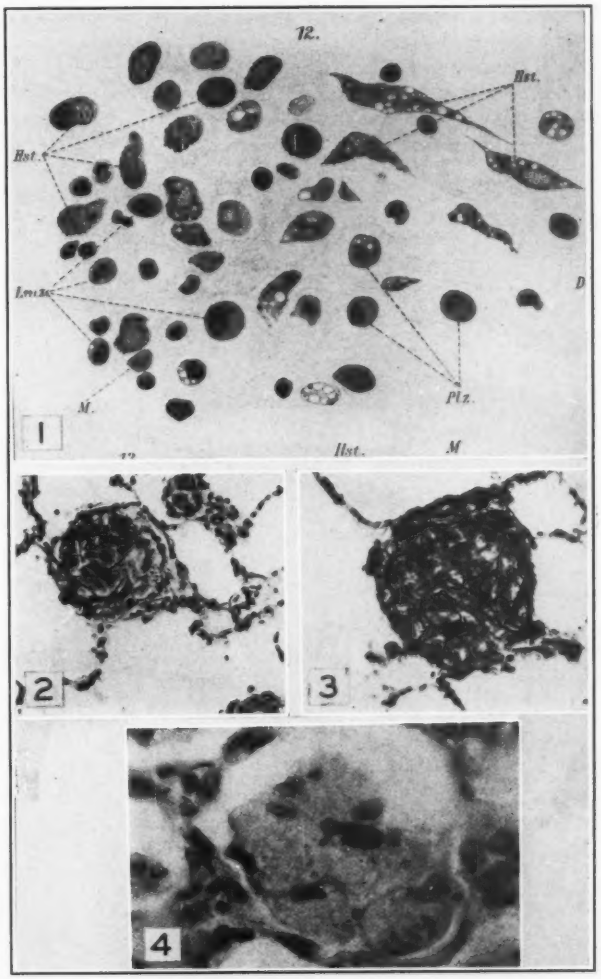

Plate 1

Fig. 1. Large mononuclear wandering cell types and variations drawn from vitally stained preparations. (From Kiyono-Die vitale Karminspeicherung, Plate III, Fig. 12. Hst-Macrophages; Lmz-Lymphoid cells; Plz-Plasma cells).

Fig. 2 and Fig. 3. The typical silicotic nodule developing within the alveolar space of the lung in its earliest stages. At this stage the lesion consists essentially of a clump of macrophages engorged with silica particles. The development of a reticulum has just begun. (From Gardner, J. Indust. Hyg., 14:18, 1932.)

Fig. 4. Pulmonary silicosis. Response of the macrophages to the presence of silica. Other inflammatory cell types do not respond to the stimulating effect of this material. (From Gardner, J. Indust. Hyg., 14:18, 1932.)

multiplication of scattered wandering cells or partially fixed cells which are indigenous to the tissues. The cells in question are (*Plate I, Fig. 1* and *Plate II, Fig. 5* and *Fig. 6*) (1) polymorphonuclear leukocytes of the neutrophilic, eosinophilic and basophilic variety; (2) macrophages, those cells which usually appear as large mononuclear elements when first seen but which change their form so frequently and so profoundly that they may be identified only by their outlandish habit of phagocytosis; (3) lymphocytes of large and small variety; (4) plasma cells.

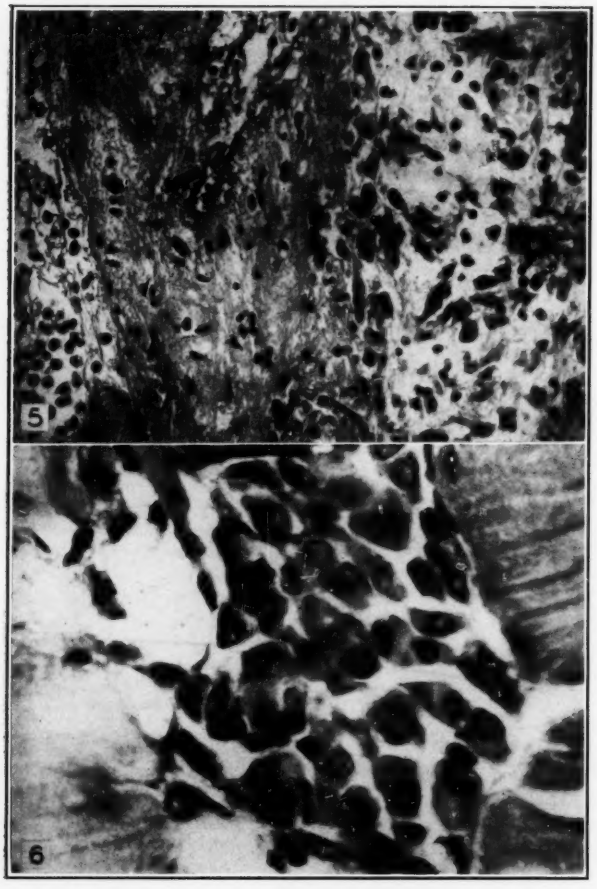

Plate II

Fig. 5. Microphotograph of acutely inflamed pleura to illustrate variation in cell types, common to all forms of inflammation.

Fig. 6. Large mononuclear macrophage reaction in degenerating voluntary muscle from a case of pneumonia. Factors responsible for the response of these cells are related to the disintegrating muscle substance which is not chemotactic for polymorphonuclear leukocytes.

In almost any inflammation (*Plate II, Fig. 5*) all types of wandering cells may be found at some time. The degree and character of protection which may be expected from the inflammatory reaction are dependent upon the play or lack of play of the factors which determine the dominant cell type since the functions of these cells are specific. A brief consideration of type reactions as exemplified in certain well-known diseases will make the problem more objective.

(a) *Acute Abscess Formation* (*Plate III, Fig. 7*).—Any agent, whether organic or of a physicochemical nature which, when introduced into the tissues kills the tissue cells, is capable of producing an abscess. Hohnfeld, in 1888, made a very careful study of the process of abscess formation, the various stages of which he listed as follows:

(1) Primary multiplication of bacteria without any immediate cellular reaction.

(2) Death and disintegration of tissue cells.

(3) Vascular congestion and dilatation.

(4) Appearance of neutrophilic leukocytes in the injured area.

(5) Appearance of mononuclear wandering cells about the periphery of the leukocytic concentration.

(6) Degeneration and liquefaction of the inflammatory mass.

The conspicuous and the essential features of this process are the growth of bacteria in the tissues, the death and disintegration of tissue cells, and the attraction to the area involved of large numbers of polymorphonuclear leukocytes. The mononuclear cells play an insignificant role and are seen in such lesions only in the advanced stages when healing is well under way.

In striking contrast to this histologic picture, consider a second common pathologic process.

(b) *Lymph Node Hyperplasia of Typhoid Fever* (*Plate III, Fig.*

8.)—Here I purposefully avoid the typhoid ulceration of the intestine in order to eliminate completely the influence upon the reaction of complicating factors such as the normal bacterial flora of the intestine. Although the lymphoid lesion in typhoid fever is usually referred to as a focal necrosis, I think that we must recognize the fact that the necrosis is usually if not always preceded by the accumulation of mononuclear wandering cells which themselves undergo degeneration. Surely one never sees a typhoid lesion dominated by the polymorphonuclear leukocyte. Obviously these cells play an insignificant role in the development of the lesion. The large mononuclear wandering cells, the macrophages, and very probably the plasma cells are the typical reactors.

A somewhat similar, if not identical morphologic response to injury, is to be seen in another, less well-known disease.

(c) *Silicosis* (*Plate I, Fig. 2, Fig. 3,* and *Fig. 4*).—The characteristic lesion in this disease consists of an inflammatory nodule in the lung which develops essentially as follows:

(1) Deposition of particles of silicious material in the alveolar spaces introduced by aspiration.

(2) Attraction into the alveoli of large numbers of macrophages.

(3) Phagocytosis of the dust particles by the macrophage.

(4) Return of the engorged wandering macrophages to the lymphatics and tissue spaces followed by their death and the depositions of silica.

(5) Fibrous tissue proliferation in response to stimulation of tissues by silica with production of nodular scarring of the lung.

In silicosis polymorphonuclear leukocytes play no part, the entire reaction being due to the mononuclear wandering cells of macrophage type.

Another most instructive and yet very complex variation in cellular response to injury is that seen in

(d) *Tuberculosis* (*Plate IV, Fig. 9*).—The development of the usual tubercle, one of the several types of histologic lesions in this disease, may be represented by the following series of events:

(1) Lodgment of tubercle bacilli in the tissue.

(2) Exudation of polymorphonuclear leuko-

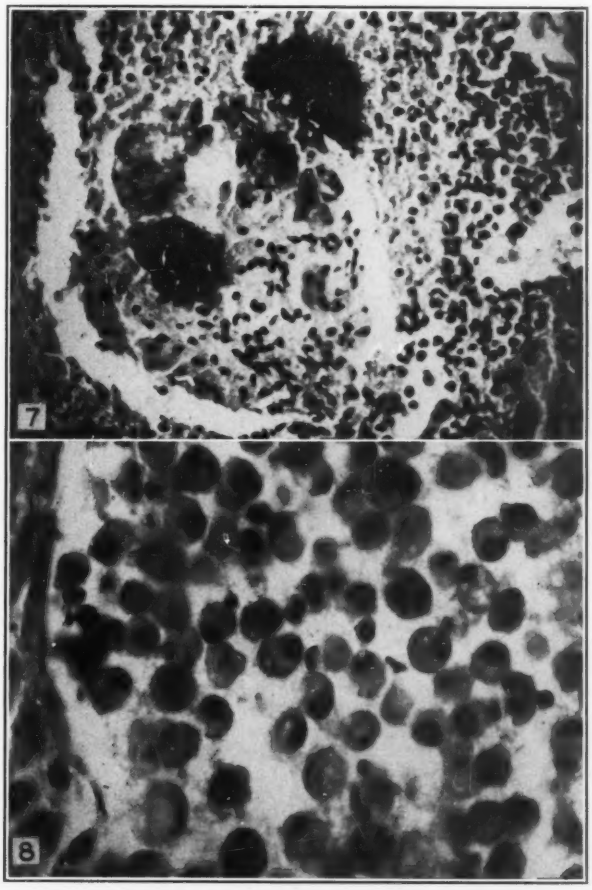

Plate III

Fig. 7. Microphotograph of kidney glomerulus to illustrate the earliest stage of acute abscess formation. Growth of bacteria has led to tissue necrosis, resulting in attraction of polymorphonuclear leukocytes only. At this stage of the lesion factors determining mononuclear cell response have not begun to operate.

Fig. 8. Typical lymph node cell reaction in typhoid fever. The microphotograph shows a lymph sinus filled with macrophages of many morphologic varieties, although essentially the same cells. Leukocytes play no part in this inflammatory response.

cytes in the injured area with phagocytosis of bacilli.

(3) Accumulation of large mononuclear macrophages with phagocytosis of bacilli and dead leukocytes containing bacilli.

(4) Transformation of large mononuclear wandering cells, the macrophages, into epithelioid cells.

(5) Formation of giant cells by fusion of epithelioid cells and/or by multiplication of nuclei and increase of cytoplasm of individual epithelioid cells.

In this procedure the part played by the polymorphonuclear leukocyte is inconstant. The predominant cell response is essentially like that in silicosis. Typical tubercle formation then appears to be a process which may begin exactly as the abscess does but which for some reason alters its course so that in the end the major role is played not by the leukocyte but by the macrophage. Tubercle formation, therefore, represents a complex cellular reaction for which, as we shall see, several specific factors are responsible.

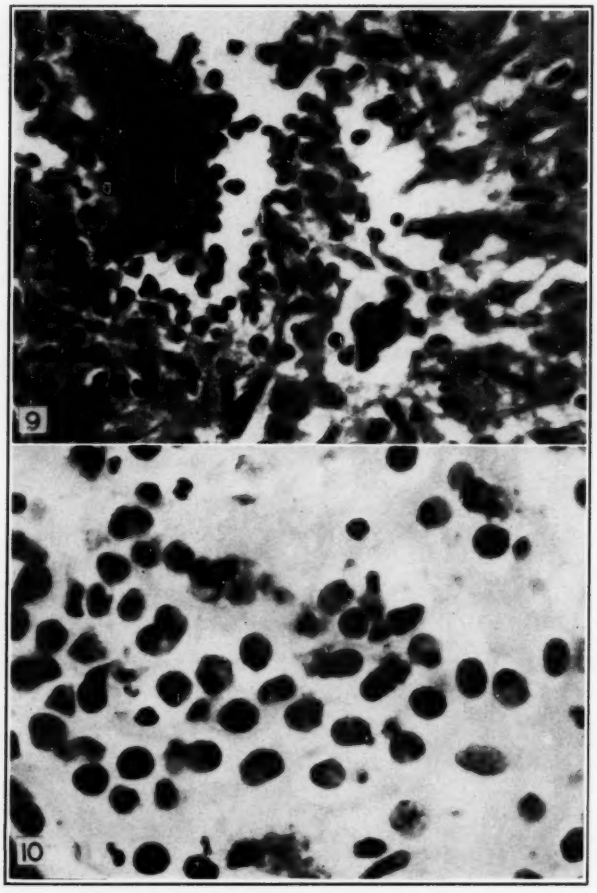

Plate IV

Fig. 9. The central area of a well developed tubercle produced by tubercule bacilli. The epithelioid cells peripherally situated and the centrally located giant cell are identical in origin and represent phases in the typical cellular response to chemical constituents of the tubercle bacillus. These cells are fundamentally the same as those shown in figures 4, 6, 8, 11, and 12, but react somewhat differently as the provoking stimulus varies.

Fig. 10. Plasma cell reaction typical of the uncomplicated syphilitic chancre. These are the cells that Goodpasture suggests may be essential as intimate hosts for the propagation of typhoid bacilli within the body.

Another interesting but puzzling cell reaction is observed in

(e) *Syphilis* (*Plate IV, Fig. 10*).—The uncomplicated syphilitic chancre is a form of inflammation characterized by the prominence of plasma cells. Although this type of wandering cell appears typically in the chancre, it does not appear exclusively so nor is it absolutely constant. Small lymphocytes sometimes predominate. The polymorphonuclear leukocyte and the macrophage are rarely seen except at the surface of ulceration. If we are prepared to accept the identity of the lymphocyte and the plasma cell, a very attractive point of view, then the cellular response to the syphilitic virus seems to be an uncomplicated one, at least in the primary lesion.

While I do not wish to bother you with unnecessary illustrative material, I cannot well ignore that exceedingly interesting group of complex cellular reactions produced by infections by certain well-known higher plant organisms. I refer especially to lesions produced by actinomyces, blastomyces (*Plate V, Fig. 11*), torula and coccidiodes. The reacting cells in all these infections are the leukocytes or the macrophages. In actinomycosis the leukocyte predominates. In blastomycosis and

coccidiosis the macrophage with its epithelioid modification usually prevails. In torula infections either may dominate the picture, although the macrophage is usually most prominent.

The striking differences between reacting cell-types in the inflammations just pictured can be explained only on the basis of the operation of potent factors specific for those cells. Since our reacting cells constitute our first line of defense against invading organisms and other harmful agents and indeed protect us from extinction, it is highly important for us to know what the factors are that govern their appearance in the tissues. Although temptation to review the details of some of the work which has been accomplished toward a discovery of these factors is considerable, I must be content to do little more than mention some of the now well-recognized factors as disclosed by recent investigations.

(a) *Polymorphonuclear Leukocyte Response.* — Almost nothing is known about the part played by the basophilic polymorphonuclear leukocyte in the inflammatory reaction. Just what factors are responsible for its appearance at the site of injury are equally mysterious. This cell is apparently unimportant if one is to judge by the number ordinarily seen in the inflammatory exudate. The neutrophilic leukocyte arrives by way of the blood stream from which it is attracted into the tissue spaces, according to the most recent work of Menkin and others, by two important factors, both of which are contained in a substance to which Menkin has given the name "leukotoxine." One of these acts upon the blood vessels and in some as yet undetermined way effects an increased permeability to both fluids and leukocytes. The other factor, the chemotactic principle, through an again unknown mechanism attracts the leukocytes toward the area of injury. The leukotoxine appears to arise in two ways, first through the disintegration of injured tissue and second through the process of bacterial metabolism. Thus the leukocytic response at the beginning of the tuberculous lesion is most probably accounted for on the basis of the presence of tissue decomposition products and need not be referred to any chemotactic influence of the tubercle bacillus itself, which as we have seen attracts especially the macrophage.

The type of cell which characterizes any inflammatory reaction is determined in the end not simply by the cells which may be most forci-

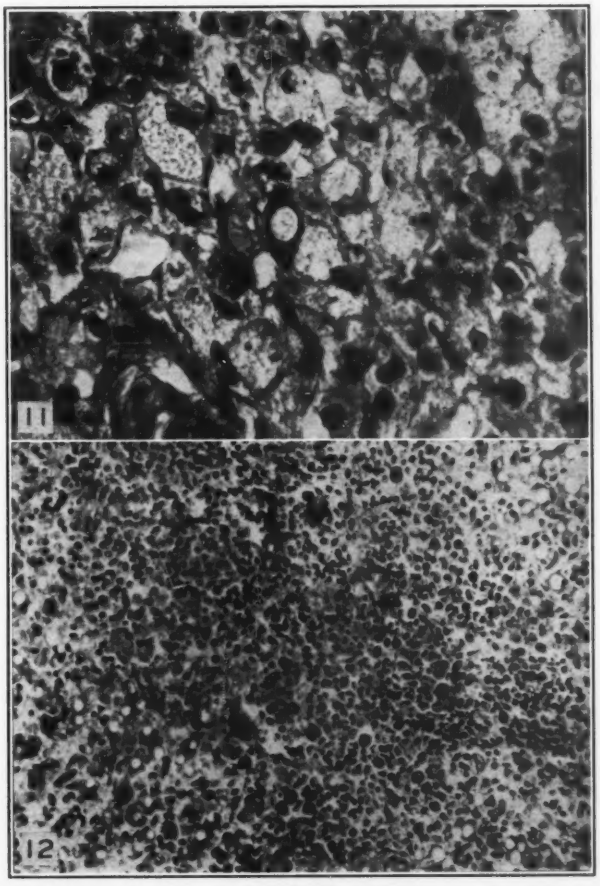

Plate V

Fig. 11. Large mononuclear macrophage response in blastomycosis. Polymorphonuclear leukocytes mingle with the huge granular macrophages, indicating the play of more than one chemotactic factor in this complex infection.

Fig. 12. Human brucellosis. This hepatic lesion is indistinguishable from that of Hodgkin's disease. The chief reacting cell is the mononuclear macrophage whose morphology varies in a pronounced way, depending upon little understood factors. Eosinophilic leukocytes and other unidentifiable mononuclear cells accompany the Dorothy Reed or Sternberg variant of the large macrophage.

bly attracted to the injured area. The cells must find an environment in which they can live and propagate. Probably one of the most important environmental factors is the hydrogen ion concentration of the tissues involved. Although some doubt has recently been thrown upon the significance of this factor, it is well known that polymorphonuclear leukocytes survive and function best in an alkaline or neutral medium, whereas the mononuclear cells appear to do best in an acid environment. If one accepts the positive significance of the tissue reaction, the character of the cellular exudate then becomes a function of the carbohydrate metabolism of the injured tissues, since the local hydrogen ion concentration is determined by tissue oxidative processes. This is about all that one may say with any feeling of assurance with regard to the polymorphonuclear leukocyte response.

(b) *Macrophage Response.*—In the case of infections by the tubercle bacillus we feel well assured that it is a constituent (phthioc acid) of the lipoid fraction of the body of the organisms that calls forth the macrophage and that this constituent is responsible for the proliferation of these cells, their formation of epithelioid cells and giant cells, and finally their aggregation and organization with the formation of the typical hard tubercle. In silicosis the macrophage reacts to stimulation by silica, another chemical agent but one utterly different from phthioc acid. It is not merely the presence of inert foreign particulate matter that leads to the proliferation of the macrophages, although it is well known that such materials when introduced into the tissues do attract these cells.

In typhoid fever the same cell responds as in tuberculosis and silicosis. At least this is true in the well developed lesion. Last year Goodpasture made the suggestion that the plasma cell also plays a very important part in the typhoid response. His studies tend to show that these cells, probably the first to respond to the presence of typhoid bacilli, are followed by the reaction of the large macrophages. The organisms appear to invade the cytoplasm of young plasma cells in the initial stage of the infection, the result of which is a form of intracellular parasitism within the body. Those plasma cells which cannot endure this experience of parasite host die and along with their included dead organisms become the stimulant for the appearance and proliferation of the large macrophages. In typhoid fever, we therefore, have another extremely interesting and highly significant factor determining cellular reaction; that is, the essential specialized cellular host requirements of an invading parasite.

Our knowledge of the factors which determine the cellular response in the various fungus infections is at best fragmentary. It is surely not enough to say that the macrophage-provoking stimulus is simply the physical character of the relatively large organisms which reside in the area of inflammations, although to be sure this is a factor of some importance. Dr. Roger Baker has been working in our laboratories for some time now on this particular problem; and while we have nothing yet to report that is helpful, we do hope eventually to know something definite about the influence of the various chemical fractions of blastomyces upon the cellular response in the very interesting inflammatory reaction produced by that organism.

(c) *Lymphocyte and Plasma Cell Response.*—So little is known of the functions of the lymphocytes both large and small, and likewise of the plasma cell, except as just described in typhoid fever that any attempt at discussion of the factors responsible for their occurrence in the inflammatory reactions, either as the primarily reacting cell as in the syphilitic chancre, or as incidental infiltrations, can be speculative only. I shall say no more about the matter than these cells react best apparently when the stimulus, whatever it be, is relatively mild and continuous over an extended period of time.

In the foregoing discussion three diseases of very different etiology have been used to illustrate the large mononuclear macrophagic reaction, namely, typhoid fever, tuberculosis and silicosis. The purpose of this selection was not only to emphasize the limited capacity of the body to react to injury but to call attention to the fact that the macrophagic reaction, one of the most primitive forms of biological response, is still extensively employed by the body for its protection under a great variety of conditions.

In contrast to this condition where widely different etiologic agents are found to provoke an identical cellular reaction is the other condition, in which very specific factors, some related to the etiologic agent and others to the host, operate to alter cell responses to one and the same agent and, therefore, produce wide variations in the character of the reaction. In this admittedly complex situation I can see certain very practical points. In the first place, because of it, critical histologic diagnosis is attended by

great difficulty and so it is, if I may refer to my original figure, that pathologic "magic" suffers great loss of face. In contrast to this rather disillusioning practical consequence is what seems to me a very optimistic and promising effect. Full appreciation of the significance of variations in morphological reaction and too, the lack of such variations, seems to me to make possible a new approach to practical therapy. I suggest that we might attempt to make greater use of the known factors which determine the variation, and by learning to control those factors, so guide the natural biological processes that their normally protective functions may become more efficient and effective.

Although pathologic anatomy may seem to have suffered some discredit in this matter, I fail to see it that way, since the pathologist's loss in diagnostic ability is more than compensated for by his increase in understanding. Through the broader concepts which result from continued experience with morphologic variations in tissue reaction and the factors which determine them, come new approaches to very old and troublesome problems. Let me illustrate what I mean by a brief reference to the work on human brucellosis now in progress in our laboratories.

Over a year ago we made the observation in the course of study at autopsy of a very rare case of fatal brucellosis that the primarily reacting cells in the tissues were those which in various combinations produce the histologic picture of Hodgkin's disease. In fact, subsequent comparative studies led us eventually to the conclusion that from the point of view of pathologic anatomy the two diseases are difficult, if not impossible, to differentiate from each other (*Plate V, Fig. 12*). Being impressed by this extraordinary similarity in morphologic reaction in the two diseases, Dr. Parsons, my assistant who performed the autopsy, was stimulated to undertake further studies which we planned to test a working hypothesis which postulated a possible close etiologic relationship between these two diseases. The first report of this work was made before the Southern Medical Association last November. At that time Dr. Parsons, in collaboration with Miss Poston, of our department of bacteriology, reported having obtained Brucella in pure culture not only from the case of brucellosis which had been studied at autopsy but from excised lymph nodes in three additional living cases, all of which had been previously diagnosed Hodgkin's disease clinically and confirmed as such by histologic studies of excised lymph nodes. Similar investigations have been carried out on all available materials since that report. As the matter stands now, a report of which was presented at the Richmond meeting of the American Association of Pathologists and Bacteriologists on the seventh of this month, ten cases of clinically characteristic Hodgkin's disease have been studied by lymph node biopsy and culture. From eight of the ten, Brucella has been obtained in pure culture. The excised enlarged nodes of the remaining two cases of the series were sterile. From a total of 33 control cases cultures of lymph nodes excised for diagnostic purposes have been positive three times. In a series of 39 control routine autopsies, cultures from numerous organs made by Miss Poston and my associate, Dr. Sprunt, have revealed the presence of Brucella nine times.

Although these observations have naturally led us to a serious consideration of the possibility of identity of Hodgkin's disease and the glandular type of brucellosis which prevails in our section, our work has not yet progressed far enough to justify any conclusion in the matter. We can say, however, with assurance that the diagnosis of Hodgkin's disease can no longer be relied upon when that diagnosis is made on the basis of both clinical and histologic studies without culture of the tissues for Brucella.

Without some appreciation of the fact of morphologic variation in inflammatory responses and some curiosity with regard to the factors which govern cell reactions, I am sure we should not now be exercised over the age-old problem of the etiology of Hodgkin's disease. To pursue this interesting problem further at this time is beyond the scope of this paper. The matter may be allowed to rest with what I have said, since our interest here lies not in brucellosis and Hodgkin's disease but in the stimulating suggestiveness of an anatomical observation of cell reactions occurring in two diseases, one of known etiology and rare and the other of obscure origin and relatively common.

RECAPITULATION

While what I have had to say does not lend itself to the formulation of specific conclusions the statement of a few generalizations may be useful.

(1) The fact that the body possesses only a limited number of mechanisms of response to injurious agents must be recognized and the significance of that limitation appreciated if we

would increase our understanding of disease and our ability as diagnosticians.

(2) Morphologic reaction constitutes perhaps our most effective, and surely our most responsive defense mechanism. It is probably also our most primitive means of protection against a universal environment which is essentially unfavorable.

(3) The general character of an inflammatory reaction is determined by many factors, some related to the host and others to the inciting agent. The cellular character of the inflammatory reaction, although ordinarily "group" constant, is subject to wide variations determined by numerous factors. Among the more important and better understood may be mentioned:

(a) The intensity of the provoking injury.

(b) The duration of the injurious stimulus.

(c) The specific physical and chemical character of the environment in which the injury occurs.

(d) The biological character of the initially reacting cells.

(e) The chemical, physical and biological character of the injurious agent.

(4) A better understanding of the factors which influence morphologic reaction in disease may be expected to influence favorably not only our efforts at rational therapy in now well-known maladies but also our attempts to elucidate the nature and the cause of still obscure disease entities.

PNEUMONIA: MEDICAL TREATMENT*

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The most important factor in the medical treatment of pneumonia is an early diagnosis. Even though this has been emphasized by previous speakers, it must be repeated with added emphasis in connection with modern treatment. The success of serum treatment is dependent upon early diagnosis and prompt typing, in order that the serum may be given during the first 24 to 48 hours, certainly not later than the third day. The same may be said of sulfanilamide, x-ray and pneumothorax. All may be used with some promise of specific action if employed early in the course of the disease.

If we are well informed as to the history of pneumonia and its treatment, we approach the bedside of our patient with a sense of wholesome humility. We must acknowledge that, until a few decades ago, the mortality rate and the essential methods of treatment remained unchanged from the time of Hippocrates in the fifth century B.C. His knowledge of the disease and his marvelous understanding of the medical management was confirmed, and continued in force by Aretaeus and Galen, his illustrious successors in the first and second centuries, A.D.

The variations in the severity of the disease and in the mortality rate, from year to year, and place to place, have often resulted in overenthusiasm for certain remedies, and, not infrequently, have given rise to unwarranted claims, even the suggestion of specific virtues. Centuries of observation and statistical studies have cleared all such claims, leaving us face to face with the record of an average mortality piercing the zigzag course of therapy with surprising constancy.

This background has been presented because it should be prominent in the mind of the physician when he first sees each case of pneumonia. With this knowledge at hand, the attending physician knows that on the average every patient developing pneumonia has 75 chances out of 100 for recovery. In other words, he must not become excited over his success until he has saved more than 75 per cent of his cases running through successive decades.

In type specific cases, antipneumococcus serum, through a trial of approximately four decades, has gained a place in our therapeutic armamentarium, the only definite cut in mortality rate in 2,500 years. Naturally, this should be our first thought when confronted with the responsibility of treatment. The efficacy of antiserum is unquestioned in Types I and II, with rapidly rising hopes in many other types, including the use of rabbit serum, now being developed for all known types. Normal conservatism punctuates the testing season with cautious reservations as to ultimate results, especially since it has been impossible to obtain in succeeding types, for which antiserum is already available, the uniformly good results experienced in the use of Type I antiserum, in Type I specific cases. Even Type II antiserum is

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much less effective than Type I. Rabbit serum should be cautiously used until its virtues and possible dangers are better known.

Above all, it is the physician's duty to make an early diagnosis, a prompt typing and to give antiserum in adequate doses the moment the presence of type specific pneumococci is discovered. A blood culture should be made in every case. It may determine the type when other sources fail. It also helps to determine the dosage of antiserum. It has been shown that the administration of Type I serum in Type I specific cases during the first 24 hours after onset cuts the mortality from one-half to one-fourth of that obtaining in untreated cases; also that the efficacy of serum is reduced approximately 50 per cent when given in the second 24 hours, and that after the third day it is too late to expect demonstrable results. There is no conclusive evidence of benefit from the use of antiserum in children. Fortunately, the mortality is low in children. In severe cases where bacteremia is present antiserum should be employed.

According to available reports, this applies to the administration of sulfanilamide. Dr. W. G. Reddick has recently reported 58 cases treated with fairly large doses of sulfanilamide during the first 24 hours, with a mortality of only 5.5 per cent. This promises results similar to those obtained with Type I antiserum. He reports a similar loss of therapeutic efficacy with the lapse of time after onset of the disease. The number of cases is too small and time of testing entirely too short to warrant giving this remedy a place with antiserum, which has been well tried.

The results of artificial pneumothorax are dependent upon early diagnosis and upon proper selection of cases. Its chief indication is in unilateral and single lobe cases, and particularly in cases with early painful pleurisy. Some recent reports tend to discredit this method of treatment. However, it may yet be found to have a valuable place in the hands of experienced operators if employed early, with careful choice of cases.

This discussion will not permit detailed instructions with reference to administration of the above methods. Such details, particularly with reference to antiserum treatment, are found in recent editions of standard textbooks on medicine. The revised edition of "Pneumonia and Serum Therapy," by Lord and Heffron, published by the Commonwealth Fund, gives up-to-date information in great detail. A careful consideration of technic, indications, contraindications and untoward results is very important.

Having recognized the undoubted specific virtues of antiserum in certain specific types, and having discussed sulfanilamide and pneumothorax because of possible virtues yet uncertain, we now come to the less glamorous though time-honored principles having to do with careful, conservative, supportive management. Emphasis must be placed upon these valuable principles for fear enthusiasm over more promising modern agents may encourage carelessness in the practice of these indispensable aids to recovery and safeguards against disaster.

As far as possible, complete physical rest should be insisted upon as soon as pneumonia is suspected. Bathroom privileges should be replaced by the bedpan and all other voluntary physical effort interdicted. The nursing should be adequate and of the best available quality. Whether graduate or practical nurses are employed, they should have definite daily instructions from the attending physician, preferably in writing. This guidance should include not only the routine instructions with reference to diet, temperature of the room, ventilation, bathing, and so on, but should include specific advice as to how best to conserve the patient's reserve energy and to save every possible strength unit. The patient should be perfectly passive in the nurse's hands, being assisted with every change of position and every move requiring muscular effort. It is the physician's duty to discuss this carefully with both patient and nurse and to make sure of their cooperation.

The various services, including medication, should be arranged and timed with the minimum disturbance of the patient. Physical examination requiring movement or turning of the patient is inadvisable unless the patient's condition suggests advancing disease or complications, such as empyema, making further investigation necessary.

The environment must be conducive to psychologic rest. The patient is entitled to an optimistic, helpful attitude on the part of physician and nurses, and must be spared the anxious and often fatalistic anxiety and sympathy of friends and relatives. Company is not to be permitted and unnecessary visitations by members of the family should be avoided.

Cool moving air through open windows cannot

be improved upon. Even cold air from out of doors is very acceptable to the majority of patients. Christian, in the last edition of Osler, recommends open air porches, the patient's bed being rolled in for needed services. Vital functions are stimulated by moving air. Variations in susceptibility to cold air and drafts must be considered and heat and ventilation regulated to meet the needs of the individual case. Nurses must always avoid the least chilling of the patient's body. When the bed covers are removed and the bare skin exposed, the room must be warm and all drafts eliminated.

Fluids, such as plain water and citrus fruit juices, should be given freely. If the patient's condition demands, fluids can be given by bowel or subcutaneously. In some cases, tea and coffee may be taken when water is refused. The intake and output should be measured and recorded. In this way, adequate amounts of fluid may be assured and kidney function observed. Patients who perspire freely require more fluids. Threatened or existing cardiac decompensation suggests a limitation of fluids. The food should be liquid, semisolid or light, and always controlled by the patient's condition and apparent capacity for it.

The bowels should be kept normally active by means of mild laxatives or enemas. It is more important to make daily examinations of the abdomen in pneumonia than to make frequent examinations of the chest. The physician can keep an eye on the abdomen without disturbing the patient. The development of tympanites may seriously embarrass respiratory and cardiovascular functions and increase toxemia. Prompt attention through diet, medication, enemata, retention of colon tube or turpentine stupes may relieve this condition. Some combination of these methods may be required. In persistent cases, posterior pituitary extract in five to eight minim doses may be advisable.

Pain, cough, nervousness and insomnia may be overcome by moderate doses of codeine. Occasionally small or moderate doses of morphine may be required, especially for insomnia or pain not relieved by codeine. If morphine is not well tolerated, chloral may be given by rectum. Loss of sleep, annoying cough and pain are more dangerous than the anodyne and sedative remedies. Some patients find a snug abdominal binder very acceptable, in that it makes cough easier, especially in case the cough is painful. In the latter case, the binder should include the hypochondriac region and restrict the flare of the lower ribs accompanying inspiration and cough. Apparently this brings about a more satisfactory function of the diaphragm and improves the circulation. Anatomical and physiologic justification for the binder restricting the lower ribs is suggested by the fact that pneumonia usually attacks the lower lobes and that ventilation of the lower lobes is brought about by the flare of the lower ribs and descent of the diaphragm. Here there is also a wide excursion of pleural surfaces and restriction of motion rests the inflamed lower lobes, restricts pleural excursion and relieves pain. Ventilation of the upper lobes is not impeded because this is dependent upon excursion of the upper five ribs.

Orthopnea also has an anatomical and physiologic phase. In normal breathing in the sitting or standing position, there is an extension of the spine during inspiration. This increases all diameters of the thorax with a corresponding increase in vital capacity. When there is a pathologic decrease in vital capacity the patient seeks the upright position because of the increase in vital capacity through this extension of the spine not exhibited in the supine position. Knowing this, we should be more tolerant of the back rest. As suggested by Meakens, we should avoid cramped positions which might unduly restrict the abdominal function in breathing.

The long-standing belief that death in pneumonia is due to heart failure has some foundation, in that the toxemia causes cloudy swelling of heart muscle, but the charge should include the whole circulatory system. Peripheral circulatory failure is one of the most dangerous developments and should be anticipated and prevented if possible. Hydrotherapy by means of cool sponging may help to prevent such a condition and offers the best means of relief when it is present. An ice cap to the head and possibly to the side or back may also be employed. When required, caffeine may be given in five to ten grain doses every three or four hours. Strychnine in large doses may be tried; grains 1/30 to 1/20 every three hours hypodermically is often serviceable. Strychnine may favorably influence circulation indirectly through increased muscle tone. In emergency, caffeine sodium-benzoate should be given. Digitalis is indi-

cated in threatened or existing congestive cardiac failure, strophanthin intravenously, grains 1/100, may be best in threatened congestive cardiac catastrophe. Peripheral circulatory failure through the influence of toxemia on the sympathetic nervous system is usually more obvious than is actual heart failure from toxic degeneration of heart muscle or from increased resistance in the pulmonary circulation.

In plethoric individuals with abrupt severe onset, bleeding may be employed with benefit. Later in the course of the disease, marked cyanosis with engorgement of neck veins may be accepted as an indication for moderate bleeding. Bleeding is not indicated in peripheral circulatory failure.

In very toxic cases threatened with dehydration, glucose intravenously is indicated. It may be given in doses varying from 300 to 500 c. c. of 10 per cent solution two or three times a day. The amount of fluids given subcutaneously and intravenously should be governed by the condition of the circulation. Disaster may follow the overloading of an already failing circulatory system.

Oxygen.—In cases of pneumonia which promise to be rather severe, oxygen should be started before cyanosis develops. It is true that cyanosis is recognized as the chief indication for oxygen and certainly its administration should not be delayed after the first signs of cyanosis appear. Nothing can be accomplished by the inhalation of oxygen after the patient becomes moribund. The tissues of the body receive their oxygen from the blood plasma and the diffuse pressure of oxygen in the plasma is the determining factor in the oxygen supply. While the blood is in the capillaries, the oxygen is transferred from the plasma to the tissue cells. This process is aided by certain chemical changes. Anything that destroys the oxygen-carrying power of hemoglobin makes oxygen administration useless unless enough oxygen is given greatly to increase the oxygen pressure in the alveoli. Oxygen-carrying power is diminished or destroyed when carbon monoxide is inhaled, also when certain drugs are given in sufficient doses to convert hemoglobin to methemoglobin, for example, acetanilid or nitrobenzol.

In pneumonia, the consolidated or blocked areas in the lung do not function; therefore they return venous blood to the tissues mixed with arterial blood, which has passed through the normal lung areas, having replaced carbon dioxide with oxygen. Soon this leads to insufficient oxygen to meet the needs of the tissue, including the nerve centers. By increasing the oxygen in the inspired air, and thereby raising the partial oxygen pressure in the healthy alveoli, not only will more oxygen enter the red cells, but some may pass directly into the plasma in the unaffected portions of the lung. This absorption into plasma helps to keep the hemoglobin saturation up, in that the plasma demands are thereby lessened.

If the heart muscle is not already profoundly affected by the toxemia, oxygen should make respiration easier and deeper, the pulse slower and fuller, and it should prevent or relieve cyanosis. It often causes the patient to be more alert and more cooperative, since it tends to overcome the stupor and mental confusion which may result from oxygen want.

Early administration is important because the want of oxygen is accompanied by an insidious progressive deterioration of nerve centers, as well as other tissues. The respiratory center is among the first to suffer. The result is rapid shallow breathing with reduction of oxygen through diminished respiratory intake. Thus a vicious circle is established and the defensive forces of the body decline.

Oxygen should be continued as long as any signs of anoxemia persist. This is to be determined not only by obvious cyanosis, but by the ashen pallor of the face which is characteristic of lingering anoxemia. If an oxygen tent is employed, 5 to 8 liters per minute may be given with a saturation of about 50 per cent in the tent. In the absence of a tent, oxygen may be satisfactorily administered through a nasal catheter. If the catheter is employed, 2 to 3 liters per minute should be sufficient. If the patient does not respond to oxygen in the percentages suggested above, more oxygen may be given. With our present methods of administration, it is impossible to raise the oxygen concentration in the alveoli above 85 per cent. Karsner has shown that under these conditions there would be no danger in giving pure oxygen for a reasonable period of time.

TREATMENT OF PNEUMONIA*

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Pneumonia is an acute infectious disease, and 97 per cent of the cases are caused by the pneumococcus, which produces a massive inflammatory exudate in one or more lobes of the lungs. That the problem of pneumonia control is receiving increased attention from medical organizations, health authorities and private agencies is strikingly evidenced by new and widespread activities in this field, both in Canada and the United States. It is more prevalent in the Northeast, Southeast and Southwest than in other parts of the country. About two years ago I began attacking this problem from a somewhat different angle, because my mortality level from pneumonia was entirely too high. In cases with high white counts that could not be explained in any other way, I had the chest x-rayed, and this revealed many pneumonias which would have gone undiagnosed. This paper is not dealing with the diagnosis of pneumonia, but I wish to say that I do not believe it is possible under the most favorable circumstances to make a positive diagnosis of this condition without the aid of the x-ray in over 85 per cent of cases. It is of great importance that an early diagnosis be made in all cases.

In the fall of 1936, we attacked our pneumonias with an armamentarium of about the following general set-up:

(1) General care.

(2) The relief of pain by small doses of opiates.

(3) Glucose 10 per cent in normal saline solution administered intravenously.

(4) Subcutaneous oxygen for cyanosis, rapid pulse, rapid respiration and delirium.

(1) The treatment of pneumonia is governed by the same general principles which apply to the treatment of all serious infections. The treatment is concerned with:

(a) The general care of the patient himself.

(b) The relief of the distressing symptoms as they may arise.

(c) The use of measures to overcome the infection.

Therefore, the first essential in the proper care of a patient is good nursing. There is probably no other disease in which the services of a competent well trained nurse who has familiarized herself with the physician's line of procedure is more valuable. It is very important to take time to explain to the nurse what you are giving and why you are giving it; it is frequently useless for the physician to write orders of which the nurse has no knowledge. Everything must be done to assure rest for the patient, and at no time should anyone be permitted to show alarm. The sick room should be well ventilated, but at no time cold, as was advocated in the treatment of pneumonia only a few years ago. Bedrooms entirely too cold were formerly contributory to the death of many pneumonia patients. This kind of treatment should be classed with the ice packs that were used in typhoid fever many years ago.

(2) For the pain of pneumonia it is entirely correct to give patients small doses of morphine of sufficient size to keep them almost, but not completely, easy. One may, if he prefers, use codeine in sufficient dosage to answer the same purpose, but beware of pushing either of these to the point of abolishing the reflexes.

GLUCOSE SALINE

(3) At all stages the patient should have plenty of fluids and I want to make this emphatic. If the average adult patient has not a urinary output of at least 1,000 c. c. in 24 hours, he is not getting sufficient fluids. We have adopted the use of 10 per cent glucose in 1,000 c. c. of saline solution, which is given intravenously every 24 hours regardless of the fluid intake by mouth. Also, from 500 to 1,000 c. c. of subcutaneous oxygen are given every 24 hours if there is cyanosis, rapid pulse up to 120, respiration up to 35, or if there are evidences of delirium. The question may be asked, why do we give the 10 per cent glucose in saline solution? The answer is this: in the first place, chlorides rapidly disappear from the body fluids in pneumonia. Second, glucose dehydrates the inflamed lung. Third, glucose is the most nutritious thing that can be used to sustain a heart in distress. Fourth, the vitality of the body cells depends largely upon the integrity of their surrounding medium, and the preservation or restoration of normal cellular environment is the sole purpose of intravenous fluid administration and represents one of the basic principles in therapy. The fluids that we are interested in are the extra-

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cellular fluids, which are readily exhausted in pneumonia, especially if the temperature is high. They are the blood plasma and the interstitial fluids. The latter, which constitutes about 25 per cent of adult body weight, is the great reservoir of the body which bathes all tissue cells. Interstitial fluids and blood plasma may be regarded as media composed of three interdependent units: water, sodium and chloride. Sodium and chloride are known as the major extracellular ions. The thing that happens in pneumonia is the rapid disappearance of these chlorides. The safety mechanisms of the body which insure stability of the cellular fluid environment depend largely upon kidney function. When the fluid intake is low, the kidneys excrete a smaller quantity of more concentrated urine. The interstitial fluid reservoir can shrink or expand in accordance with body needs. However, there are limits beyond which the stabilizing mechanisms cannot go, and these are reached in the pneumonias, with their high temperatures, if fluid is not adequate.

A small fluid volume intake results in dehydration with all of its toxic symptoms.

SUBCUTANEOUS OXYGEN

(4) What may we offer the pneumonia patient with cyanosis, rapid pulse, rapid respiration and delirium? Oxygen.

Oxygen is the most abundant of all the elements and is essential to all forms of life, yet strange to say it was discovered only a little over 160 years ago by Joseph Priestley. During the past ten years a great amount of literature has appeared on oxygen therapy, especially the concentration inhalation type, using tents and oxygen chambers. It is unfortunate that oxygen therapy by this method can be afforded only by the very wealthy. For some unknown reason very little subcutaneous oxygen has been used, although this is the most efficient and the least expensive method of employing it. By this method such a small amount of oxygen is used that the cost of the gas is negligible. Oxygen, when given subcutaneously, is readily diffused from one tissue to another. It is taken up by the blood wherever available and distributed to all parts of the body. In the administration of subcutaneous oxygen it is necessary to have proper control and regulation of the gas pressure and measurements of the amounts given.

Subcutaneous Versus Inhalation.—The reason that I prefer the subcutaneous method to the inhalation is the fact that many times the alveoli of the lungs are obstructed and the patient does not get oxygen by inhalation, whereas by the subcutaneous method he cannot help but absorb it. In other words, one feels sure that by the subcutaneous route it will reach its destination.

The primary purpose of oxygen therapy, whether inhalation or subcutaneous, is the combating of anoxemia. The clinical importance of this has been recognized for many years and the time will soon come when the degree of anoxemia in certain diseases will be considered as regularly as the leukocyte count is now considered in appendicitis. The oxygen content of the blood is rarely of routine concern in the minds of the majority of physicians, because of the fact that knowledge of anoxemia is relatively newly acquired and certainly complex. The method of determining the oxygen saturation of the blood needs to be simplified. The most practical test for determination of anoxemia is by the injection of two or three hundred c. c. of oxygen subcutaneously. If there is anoxemia, the oxygen is rapidly absorbed with marked relief of symptoms. The injections and amounts required can be repeated as necessary to relieve the condition. I have not used an oxygen tent, but it is my information that the gas is given in these at a concentration of 60 per cent or higher. Common sense reasoning leads me to the conclusion that at this concentration it is not entirely harmless to already inflamed lung tissue, thereby nullifying the expected benefits.

The most remarkable feature of the subcutaneous method is that injections of small amounts of oxygen are very efficient. It seems unbelievable to one who knows what large amounts of oxygen are required by the inhalation method to consider that oxygen deficiency is really rather minute and can be easily supplied by the subcutaneous injection of the gas. The amount of deficiency is in proportion to the difference between the supply and the requirements, and it is usually small. It is like the old fable of the last straw that broke the camel's back. This "last straw" often comes in pneumonia cases when just a little might have carried the patient over.

Dosage.—The average dose of oxygen, when given subcutaneously to an adult, is 500 to 1,000 c. c., repeated as often as it is absorbed. Absorption can be easily ascertained by crepita-

tion. The average pneumonia patient would be quite comfortable on one or two injections per day. It will be necessary to give it more often if there is much cyanosis or air hunger. The distress and anxiety of these poor patients are markedly relieved and they feel much easier, until their oxygen is again exhausted and their dyspnea begins to return. Then of course the injections should be repeated.

Indications.—Oxygen therapy is indicated in all pneumonias which are complicated by symptoms of anoxemia, asphyxia or rapid breathing; in pneumonias with delayed resolution or in those with very little or no expectoration. Oxygen administration is compatible with any and all kinds of treatments. It was used first in France. It was then very slowly accepted in Germany, but finally took its place. And it is my belief that in the near future subcutaneous oxygen therapy will be considered as important when it is required as fluids are considered today in the dehydrated patient. When the importance of the action of asphyxia on the body cells is more thoroughly appreciated, oxygen will be freely used. Unless one is burned alive, the tissues of the body always die of asphyxia. It is evidently asphyxia of the brain cells that causes the pneumonia patient to become unreasonable, fretful and mentally confused or delirious. The relief of his mental condition is merciful and invaluable, and it alone justifies the use of subcutaneous oxygen.

Dr. Raymond reports that he has repeatedly seen subcutaneous injection of oxygen produce veritable resurrections. Dr. Melchior, of Germany, reports having given more than twenty-five hundred injections without having had any complications; the amounts he reports having used are from one-half litre to as much as eight litres at one injection.

T. S. Kirk, in a British medical journal, reports administration of subcutaneous oxygen to 200 cases without any complications whatever. He says that it is a perfectly safe procedure.

It is a vital feature, of course, in a case of pneumonia with respiratory distress to secure rest, and to permit the patient to be comfortable, thereby saving him from wearing himself out struggling for breath. Oxygen is the sovereign remedy for anoxemia and it is my opinion that the subcutaneous route of giving it is the most effective as well as by far the most economical.

Site for Injection.—We have found from experience that a point for injection just posterior to the midaxillary line is the most satisfactory site, because if there is any gravitation it will be of no consequence whatever. The reason I mention this is the fact that if it is injected too far anteriorly it may occasionally gravitate under the skin of the neck and cause innocent pressure symptoms.

ADVANTAGES OF THE SUBCUTANEOUS METHOD

(1) Certainty of absorption.

(2) It is simple and easily given and may be given by a flashlight in any home.

(3) The equipment is simple and may be easily carried to any home.

(4) The economical side is quite a consideration.

SULFANILAMIDE AND ALLIED PREPARATIONS

At the meeting of the American Medical Association in Atlantic City in 1937 the use of sulfanilamide was introduced to the American physician and in the fall of 1937 we began to give two of these preparations in connection with our treatments as above outlined. We soon definitely decided that "neo-prontosil" (disodium salt of 4-sulfamido-phenyl-2'azo-7'-acetylamino-l'hydroxynaphthalene-3':6' disulfonic acid) was far more effective in pneumonia than sulfanilamide because of the fact that the response to it was quickly achieved and this drug was very definite in its action in pneumonia. The number of cases treated in this series is 112 with four deaths, a mortality of a little over 3.5 per cent. I am aware of the fact that this series is too small in number to establish definite treatment. But one is led to believe this combination of treatments may prove to be more effective than anything thus far used.

SUMMARY

After many variations of the dosage and spacing of "neo-prontosil" injections we found the best average dose for us to use was 15 c. c. 2½ per cent every six hours. We tried the drug out on an eight-hour schedule and found that it did not control temperature.

(1) The average amount of "neo-prontosil" given to each case in this series was 100 c. c.

(2) The average time after administration before the temperature began to fall was 30 hours. Four and one-half days were required for a return to normal.

(3) The average temperature before administration of the drug was 103.6°.

(4) The average pulse rate was 116.

(5) The average respiration was 33.5.

(6) The average age in these cases was 32.8 years.

(7) The youngest case in this series was 10 months old.

(8) The oldest case in this series was 86 years old.

DISCUSSION (*Abstract*)

Dr. R. A. Miller, San Antonio, Tex.—Dr. Yeiser's experience with subcutaneous oxygen therapy is interesting and commendable. Anything to relieve the dyspnea of pneumonia patients is welcomed by all physicians. Our experience with this method is limited. The Pediatric Department of the Robert B. Green Memorial Hospital used it in 1935, but has since discontinued its use. Their chief reason for preferring the oxygen tent or nasal catheter is that the oxygen tension of the blood can be elevated and maintained at the normal amount satisfactorily. The comparatively small amounts used with the subcutaneous method are not apparently sufficient to control dyspnea and cyanosis. The oxygen tent is expensive, and as Dr. Yeiser has mentioned, subcutaneous oxygen therapy is cheap, but when a life can be saved its expense should not be considered. In telling the patients very bluntly, we explain that the expense of an oxygen tent does not compare to that of a funeral.

Fortunately in San Antonio we do not have to bother with the expense, as a surgical supply house furnishes our oxygen tents. When a tent is prescribed as treatment, the patient's ability to pay is not considered. Naturally many patients cannot afford to pay, but the company gratefully considers this their courtesy to the profession and its patients.

From an anesthetist's standpoint, it is interesting to follow the trend of the use of oxygen as a treatment over the past few years. In 1929 in Grace Hospital, Detroit, Michigan, an internist was using oxygen subcutaneously for the experimental treatment of diabetes mellitus. More recently I heard of a physician's using oxygen carbon dioxide and oxygen inhalations to inhibit the progress of cancer. I am not criticising wayside experimenters, for some day they may discover a very valuable remedy.

Because the subcutaneous oxygen method is simple, inexpensive and easily administered, I think more work should be done to prove its worth, especially on the line of study of the blood oxygen tension.

THE TREATMENT OF PNEUMONIA IN INFANCY AND CHILDHOOD WITH SULFAPYRIDINE*

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Domagk,[1] in his original publication on the use of "prontosil," indicated that this dye had some effect upon pneumococci. However, the effect was not great enough to warrant its use in all types of pneumonia. It remained for Ewins and Phillips, in England, to prepare 2 -(p-amino-benzene sulfonamide) pyridine, best known today in the United States as sulfapyridine.

The first article to appear in the literature on the use of sulfapyridine in pneumococcic infections was that of Whitby[2] in 1938. He reported that sulfapyridine was chemotherapeutically active in experimental pneumococcic infections in mice and that it had a low toxicity for animals. Fleming[3] presented evidence that sulfapyridine *in vitro* increased the killing power of human blood serum toward pneumococci. Telling and Oliver,[4] in June, 1938, reported a case of Type III pneumonia with massive collapse which recovered under sulfapyridine therapy.

Evans and Gaisford,[5] in July, 1938, treated with sulfapyridine alternate cases in a group of 200 pneumonia cases. Their mortality was 27 per cent for the control group and 8 per cent in the sulfapyridine group. The only toxic effects observed were cyanosis with methemoglobinemia, nausea and vomiting. No subsequent report has had a greater mortality than 8 per cent. The same authors reported the first 40 cases of childhood bronchopneumonia treated with sulfapyridine. There were 2 deaths due to pneumococcal peritonitis. They observed no toxic effects in children. Their dosage schedule was as follows:

1-3 months	0.8 grams daily
6-24 months	1.5 grams daily
3 years	2 grams daily
5 years	3 grams daily

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Flippin and Pepper[6] reported the first 2 cases in the American literature, both of which recovered. Dyke and Reid,[7] in November, 1938, reported 8 cases, all of which recovered with immediate fall of temperature and pulse rate with rapid clinical improvement.

During 1939, many additional reports in the British and American literature have appeared. Meakins and Hanson[8] reported 30 cases with a 3 per cent mortality. Agranat, Dreosti and Ordman[9] reported 100 cases of definitely diagnosed lobar pneumonia treated with sulfapyridine with no deaths, while there was a 4 per cent mortality in a control group. They noted a shorter pyrexial period, a milder course without any toxemia, and a reduced period of convalescence as compared with controls. These results follow closely all the previous reports. Graham and associates[10] report 50 cases with a 6 per cent mortality. Blood sulfapyridine determinations were directed towards maintaining an average concentration of 7-8 mg. in adults. In all their cases the temperature fell in 36 hours. There were 2 cases of empyema which recovered with the use of sulfapyridine and thoracentesis. They had a case of granulocytopenia with granular cells down to 8 per cent, but the count returned to normal when the drug was discontinued. Nausea was present in 66 per cent of cases and vomiting in 30 per cent.

Barnett and associates[11] were first to report on sulfapyridine in childhood pneumonia in the United States. They used the dosage of Evans and Gaisford, giving the drug in powdered form, in water, milk, or fruit juices. Their results in 40 cases were just as encouraging as the previous reports. Their blood level of sulfapyridine was 5-10 mg. They quote Whitby[12] to show that sulfapyridine is rapidly absorbed and is excreted more slowly and that the maximum concentration is obtained in 12 hours. Their toxic effects were similar to those already mentioned. For cyanosis they used intravenous methylene blue with some success. They had a case of agranulocytosis, which died later of a fatal hemorrhage after the white blood count had returned to normal. They called attention to a report of 2 cases[13] of agranulocytosis in the British literature.

Flippin and associates[14] reported 100 adult cases with 4 deaths, 2 being Type III. They remarked on the ability of sulfapyridine to cause a critical drop in temperature in 24-48 hours followed by prompt clinical improvement. They had no empyema. They reported the usual nausea and vomiting, 1 case of generalized morbilliform rash, 1 acute hemolytic anemia, 1 leukopenia, and 1 case of drug fever. All of these complications cleared rapidly when the drug was stopped.

MacColl,[15] in March of this year, using the dosage of Evans and Gaisford, reported 33 cases of childhood pneumonia. He observed the effects already noted by previous authors, especially the rapid fall in temperature and the early clinical improvement. MacColl says that a concentration in the blood of children of 2 mg. per cent is therapeutically adequate. The usual toxic manifestations were observed.

Hodes and associates,[16] in April, 1939, reported 33 sulfapyridine-treated cases of primary pneumonia and 38 cases of pneumonia complicating measles, all of which recovered. Five of their cases had positive blood cultures. Type XIV pneumococcus was observed most frequently in younger children and Type I in older children. The dosage used was 1-6 grams in 24 hours, depending on age. The drug was continued until the temperature had remained normal for 5-8 days. The clinical course was similar in all age groups. Regardless of prior duration of the pneumonia, recovery took place in the same time after therapy was begun. The early clinical improvement and fall in temperature were observed, but consolidation did not disappear more rapidly. Recurrence occurred in 2 cases where the drug was stopped too soon. No empyema occurred and only 3 cases of otitis media. There was 1 generalized morbilliform rash with which fever occurred, but subsided 8 hours after the drug was stopped.

Wilson and associates,[17] in April, 1939, reported 70 cases of childhood pneumonia, of which half received sulfapyridine and half did not. No fatality occurred in either group. The drug shortened the course of pneumonia by 3-4 days and improvement began earlier in the sulfapyridine-treated cases. Two relapses occurred when the drug was stopped and 2 other cases were uninfluenced in their course by sulfapyridine. They believe a blood concentration of 4 per cent is adequate in children. They used sodium bicarbonate with sulfapyridine. Their dosage was 1-1½ grains per pound of body weight every 24 hours with a large initial dose. The usual toxic effects were seen. A decreased number of cases of suppurative otitis media were

seen plus a slight decrease in other suppurative complications.

In May, 3 cases[18] of hematuria were reported, 1 being gross hematuria. Two of the patients had severe abdominal pain of renal or ureteral origin with nitrogen retention due to renal insufficiency. These signs all cleared when the drug was discontinued and fluids were forced. In June, a report[19] of 50 adult cases gave similar results to those already mentioned.

We are here reporting a series of 40 cases of pneumonia, in infancy and childhood, treated with sulfapyridine during the period from December 31 to May 1, 1939. We contrast these with our cases during the same period for the preceding year when only supportive and symptomatic treatment was available. In the last month of 1938 and the first three months of 1939, sulfapyridine was difficult to obtain in adequate amounts and we had in this period, in addition to the 40 sulfapyridine-treated cases already mentioned, 25 cases which were not treated with sulfapyridine because of an inadequate supply of the drug. Owing to a very limited and uncertain supply of sulfapyridine, the early cases in the series received an inadequate amount in some cases and in others the use of the drug was instituted very late.

The age group of the 40 cases was between 2 months and 12 years of age and divided evenly as to sex. In *Table 1* can be seen the anatomical types of pneumonia treated, of which 31 were confirmed by x-ray.

Table 1

Type of Cases	Number of Cases
Bilateral bronchopneumonia	8
Unilateral bronchopneumonia	10
Left lower lobar pneumonia	10
Right lower lobar pneumonia	6
Right upper lobar pneumonia	3
Left upper lobar pneumonia	1
Unclassified lobar pneumonia	2
Total	40

The pharyngeal swab method of obtaining material for Neufeld typing was used in all cases. In many cases pneumococci were observed, but no type was found. *Table 2* gives the type of pneumococci which were typed.

Our dosage of sulfapyridine was as follows: in children under 6 years of age 7½ grains were given as the initial dose and 3¾ grains every 4 hours thereafter until the temperature was normal and clinical improvement was observed,

Table 2

Types of Pneumococci	Number of Cases
Type XIV	3
Type VI	2
Type XIX	1
Type VII	1
Type IV	1
Type I	2

following which the dose was decreased gradually over a period of 3-5 days. In children over 6, 15 grains were given as the initial dose and 7½ grains thereafter until a fall in temperature and improvement occurred, when gradual reduction of dosage was carried out. Our average total dose of sulfapyridine was 80 grains; the maximum dosage in any one case was 150 grains; the minimum dose in any one case was 15 grains.

The average duration of the pneumonia before admission to the hospital was 2½ days. Maximum duration before admission was 7 days and the minimum 1 day. In 30 cases, sulfapyridine was started within 24 hours of admission to the hospital. In 5 cases it was started within 48 hours; in 1 case in 72 hours; and 4 cases longer than 3 days. The delay in starting sulfapyridine was due chiefly to inadequate supply earlier in the year. In 27 cases improvement began within 24 hours of the beginning of sulfapyridine therapy. The temperature returned to normal and the patient remained afebrile thereafter. With the fall of temperature to normal, marked clinical improvement was observed, with decrease of pulse rate and respiration toward normal levels and with disappearance of lassitude, headaches, irritability and other toxic manifestations. In 4 cases improvement began 48 hours after the drug was started and in 7 cases it began in 48-72 hours. In 2 cases improvement occurred later than 72 hours after the institution of therapy because the drug was discontinued too soon. The patient had recurrence of symptoms. The average duration of illness as measured from the beginning of therapy to beginning resolution of physical signs and complete absence of fever was 3½ days.

In our control series, from the same period of time in the preceding year, there were 27 lobar pneumonias, of which 19 were males and 8 females, and 30 bronchopneumonias, of which 17 were females and 13 males. All were treated symptomatically. X-ray confirmation was obtained in 13 cases. *Table 3* shows the anatomical distribution. The age group was 6 weeks

to 10 years. Typing from a pharyngeal swab by Neufeld method was done in most cases, but only 2 were typed, one a Type I and one Type XIV.

Table 3

Type of Cases	Number of Cases
Left lower lobar pneumonia	14
Right lower lobar pneumonia	9
Bilateral lobar pneumonia	3
Right upper lobar pneumonia	1
Bilateral bronchopneumonia	5
Unilateral bronchopneumonia	25
Total	57

The average duration of illness before admission in this control group was 3 days, the minimum 1 day, and the maximum 10 days. Treatment was supportive, consisting of forcing fluids, tepid sponges, stimulants as needed, and oxygen occasionally. Treatment was started on all cases on admission. Improvement began on the average 5 days after the onset of treatment. The average duration of illness, as characterized by fall in temperature to normal plus beginning resolution with disappearance of physical signs, was 6½ days. Contrast this with 3½ days in the sulfapyridine-treated cases. In the control series there were 8 deaths, 4 being lobar pneumonia and 4 bronchopneumonia. The pathologic condition in 2 of the deaths was confirmed by autopsy.

Complications in the control series included 7 cases of suppurative otitis media, with one mastoid which was successfully operated upon; 3 cases of empyema, 1 of which died, 1 had a thoracotomy and recovered, and 1 recovered with a repeated thoracentesis.

In the sulfapyridine cases, there was only 1 death, not included in the total of 40 cases reported, which occurred in an 8-months-old infant admitted 2 days after the onset of illness with a bilateral bronchopneumonia and a temperature of 105°. At the time the child was admitted, sulfapyridine was not available and the drug was not started until the day of death, when the patient received 50 grains in 16 hours, but the child was already moribund and suffering from a peripheral vasomotor collapse. Autopsy revealed a massive bilateral bronchopneumonia.

Complications in the sulfapyridine-treated cases were as follows: 1 case of empyema in a bilateral bronchopneumonia, but sulfapyridine was not started until after the empyema appeared, and when a hemolytic streptococcus was isolated sulfanilamide was used plus repeated thoracentesis, with complete recovery.

Blood concentrations of sulfapyridine were obtained in 19 cases, mostly during the later part of the series, because during the early period of use of this drug, the technic of estimation of blood concentration was unsatisfactory and as a consequence the determinations were likewise unsatisfactory. In the later months with the development of a proper technic, the estimations became entirely satisfactory. Concentrations were obtained only in the older children and therefore the number of cases with blood determination is relatively small. Concentrations were maintained in two-thirds of the cases at a level of between 5 and 9 mg. per 100 c. c. The maximum concentration obtained in any single case was 12.5 mg. Concentrations were obtained every other day for the first few days and when the drug was decreased in dosage no further concentrations were obtained. In a case which received 112 grains of sulfapyridine, a concentration could be built up in the blood, although the case responded in the usual way.

Blood counts were taken every day for the first 4 or 5 days and every second or third day thereafter. We observed 2 cases of leukopenia of an agranulocytic type. In 1 of these the white blood count fell to 2,500 on the third day of administration of sulfapyridine. The total count was normal in 24 hours after cessation of therapy, although granulocytic depression was still present. In our series of cases, we observed a characteristic granulocytic change in the differential blood picture. Usually in most cases treated with sulfapyridine, the total blood count fell as the temperature fell. With this fall in the white count, there was seen a decrease in the granular cells and an increase in the lymphocytes and monocytes. The lymphocytes in 2 cases exceeded the granulocytes. In the series of 40 cases there were 22 with this change in the differential picture. When the drug was stopped the blood picture gradually returned to normal. In the 22 cases the percentage of both types of cells tended to become equal; for example, a case had 52 per cent granulocytes and 47 per cent lymphocytes 48 hours after the onset of therapy. This shift was observed to begin within 24 hours after the drug was started, as blood counts taken before beginning sulfapyridine showed the usual increase in segmented cells characteristic of pneumonia. In some of the 22 cases, the shift was gradual

and in others more rapid. In a case of bronchopneumonia with a bilateral suppurative otitis media, the white count rose from 13,000 to 45,600 in 24 hours after sulfapyridine was started, and in 24 hours after the drug was discontinued the count was back to 10,200, but showed granulocytic depression. In *Tables 4* and *5* are demonstrated the changes in the white blood count and differential pictures of the 2 cases of leukopenia from the date of admission until the white blood count had become stabilized. It will be seen that in the second case of leukopenia the white blood count fell to 3,800. In both cases, as soon as sulfapyridine was discontinued the total white count rapidly returned to normal, although granulocytic depression still persisted.

In only a few cases was there a drop in hemoglobin and red cells and this was not marked. We had a case of hematuria, which was never visible grossly. Daily urines were obtained in most cases while sulfapyridine was given, and in this case on the second day after the drug was started, red blood cells too numerous to count were reported and again the next day. The drug was stopped and the urine was free of red blood cells thereafter. There was no cyanosis attributable in any of our cases to sulfapyridine. Two cases were cyanotic on admission and were given oxygen and sulfapyridine with disappearance of the cyanosis. We had a case of a generalized morbilliform rash which cleared 36 hours after the drug was stopped. Nausea and vomiting were very rare and not attributable to the drug. Children were acidotic on admission and as soon as the acidosis was overcome, vomiting ceased. In only one case was the vomiting related to the use of the drug and in this case after one dose it had to be dis-

Table 4

Date	Total W.B.C.	Stab.	Segm.	Lym.	Mono.	Eos.	Bas.	Irritation
4/17	5,900	0	77	19	4	0	0	0
4/18	5,000	0	62	26	12	0	0	0
4/19	3,800	3	58	24	15	0	0	0
4/20	3,950	4	48	37	7	1	0	3
4/21	5,300	2	24	49	3	5	Prolymphocytes 16 Lymphoblasts 1	
4/24	5,560	1	41	52	2	1	1	0
4/25	6,650	1	35	52	9	3	0	0
4/26	5,250	0	35	56	4	4	1	0
4/27	5,000	0	40	59	1	0	0	0

The blood concentration of sulfapyridine in this case was never more than 1.9 mg.

Table 5

		Juv. Stab.							
3/25	14,100	4	9	71	16	0	0	0	0
3/27	8,350	1	2	62	34	1	0	0	0
3/28	2,500	0	1	55	40	3	1	0	0
3/29	4,750	0	0	62	29	7	1	1	0
3/30	4,150	0	1	50	42	4	3	0	0
3/31	4,700	2	2	58	33	3	2	0	0
4/1	6,500	0	2	61	33	4			0

The blood concentration of sulfanilamide was never more than 8.3 mg.

continued. After the drug was discontinued, empyema developed and thoracotomy was performed, with recovery thereafter. Since sulfapyridine was not given because of the vomiting, this case was not included in our series of cases.

In two cases previously mentioned, sulfapyridine was discontinued too soon. After a typical response to it, relapse with fever and signs of toxicity occurred. The drug was then again given with the usual effect and rapid recovery.

CONCLUSIONS

(1) Sulfapyridine has been found to be an exceedingly valuable agent in the therapy of both lobar and bronchopneumonia in infants and children, regardless of the type of pneumococcus present.

(2) Sulfapyridine causes a rapid fall in temperature and pulse rate with early clinical improvement, shortening the acute stage of the pneumonia.

(3) In the reported series of cases, we had no deaths as compared with 8 deaths in the control group.

(4) Suppurative complications of pneumonia such as empyema and otitis media and other pyogenic secondary conditions were markedly decreased by the use of sulfapyridine.

(5) We had two cases of agranulocytosis which recovered as soon as the drug was stopped. A tendency to granulocytic depression of the white blood cells was observed in 22 cases. However, with the cessation of the drug, the blood picture returned to normal.

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EARLY ROENTGENOLOGIC SIGNS IN SOME OF THE MORE COMMON DISORDERS OF THE HIP*

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The hip is affected by disease no more frequently perhaps than any other joint of the ball and socket or hinge type, but because of the great disability resulting from a comparatively small amount of disease process, the early recognition and diagnosis of any disorder is of great importance. A great deal has been written concerning hip disorders, and many pathologic processes have been described. We have not attempted to go into any great detail, but have confined ourselves to giving a brief summary of the early roentgenologic signs of some of the more common disorders, carrying the process only to the stage where a diagnosis can be made with a reasonable degree of accuracy. A brief review of the pathology and clinical signs has been included, in some cases, to give a more comprehensive picture of the process. The disorders encountered most frequently are: (1) congenital dislocation of the hip, (2) osteochondritis deformans juvenilis (Legg-Calvé-Perthes' disease), (3) slipping of the upper femoral epiphysis (epiphyseolysis), (4) tuberculosis of the hip, (5) acute pyogenic osteo-arthritis, and (6) degenerative arthritis (hypertrophic, osteo-arthritis). Another disorder, coxa magna, has been included, not because of its frequency, but because it has been considered by some to belong in the same category as osteochondritis deformans juvenilis, and slipping of the upper femoral epiphysis.

Congenital dislocation of the hip is rarely

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diagnosed before the patient is one year of age because, as a rule, nothing abnormal is noted until the infant begins to stand and walk. The normal newborn pelvis is roughly triangular in shape with the base above and the apex at the pubis. The sides are approximately straight, with no inward angulation in the region of the acetabula. Once the child has begun to walk, the upward thrust of the femur causes the acetabula to be pushed in slightly. At the same time, the downward pressure of the body causes flaring of the ilia, the net result being a triangular-shaped pelvis with a slightly broadened base, and sides slightly curved, the convexity inward.[1] The congenitally dislocated hip presents quite a different picture from the normal. There is no inward angulation on the affected side in the region of the acetabulum; the acetabulum is shallow and the superior portion of the rim shows poor development, giving a sloping roof;[1 2] the neck of the femur is frequently distorted and smaller than normal. The trochanter on the affected side is usually high, since the dislocation is usually upward,[1] with the epiphysis at varying distances from the acetabulum, but never in it. The neck of the femur often runs more nearly vertically instead of forming the normal obtuse angle with the shaft of the femur. In case of a bilateral dislocation, the above findings are present on both sides, though the changes may be more marked on one side. The diagnosis in infants before ossification centers have formed in the femoral head is apt to be difficult, but Norrison[2] reports that the socket on the affected side is long and narrow, the portion above the Y epiphysis being slightly elongated and much more shallow than normal, and that the femoral head, as determined by the diaphyseal portion of the femur, is above the Y epiphysis instead of on a level with or below it. This condition must be differentiated from traumatic dislocation, in which there are a well developed, normally shaped pelvis, acetabulum and femur and tuberculous and nontuberculous epiphysitis, in which are found evidence of bone destruction and new bone formation in the head of the femur, or acetabulum, or both.[1] If the child is old enough, there is also evidence that weight-bearing causes changes in the pelvis. Occasionally malunion of fracture of the femoral neck with fibrous or cartilaginous union will give the clinical picture of congenital dislocation, but the roentgenologic picture readily makes the correct diagnosis.

Osteochondritis deformans juvenilis (Legg-Calve'-Perthes' disease) shows clinically much the same picture as tuberculosis of the hip. The patient develops a limp, and about 50 per cent complain of some pain in the affected hip. The intensity of the pain is variable in different patients, ranging from a slight ache to severe pain, producing the so-called "night cries." On examination the hip is usually found to show some degree of fixation by muscle spasm, usually in adduction, with some limitation of motion in all directions. The greater trochanter is somewhat prominent, and there may be some slight muscular atrophy, but practically never is there much tenderness, heat or swelling of the hip region.[3 4]

Several theories have been advanced as to the etiology of this disorder, but the exact cause has not been definitely established. Howorth and Ferguson[4] state that circulatory disturbances in or adjacent to the femoral head are the cause. These disturbances may result from inflammation in the joint, as a subacute infectious arthritis; inflammation or tumor adjacent to the joint; fracture of the femoral neck, or traumatic slipping of the epiphysis; trauma to the femoral head, or tension of the soft tissues about the femoral neck, seen especially after reduction of congenital dislocations. The first two conditions produce sclerosis and scarring of the tissues about the neck and head, acting to cut down the blood supply, the last condition acts in a purely mechanical manner to produce the same result. This theory is in accordance with experimental findings reported by Miltner and Hu.[5] They succeeded in producing gross and microscopic changes in the hips of dogs and rabbits similar to Perthes' by stripping the periosteum from the femoral neck and ligating the round ligament, thus interrupting practically the entire blood supply to the head. The net result was an aseptic necrosis of bone, with flattening due to weight-bearing. Other theories advanced are the developmental theory of Murk Jansen and the infective theory of Phemister, neither of which has been definitely substantiated by experimental or clinical and surgical observations.

The pathologic changes in the very early cases, as reported by Ferguson and Howorth,[4] consist of thickening, edema and chronic inflammation of the synovial membrane, periosteum and joint capsule, with no changes in the joint cartilage. In later cases, they report that the synovial membrane and periosteum are scarred, inelastic and thin, with extensive narrowing of the lumen

of vessels. The bone pathology in a well advanced case consists of massive subchondral bone and marrow necrosis, with granulation tissue forming around the necrotic bone, later forming and being replaced by osteoid tissue and new bone. The necrotic head may show compression fractures.[5,6] The joint cartilage is always preserved, though it may become edematous and fibrillar[5] and often shows proliferation at the margins.[4] The epiphyseal line may be intact, interrupted or destroyed by the pathologic process.[5,6] Similar bony and cartilaginous changes may occur in the acetabulum, but are not common, nor as extensive.

The x-ray findings are characteristic of the disorder. In the incipient stage, as reported by Waldenstrom,[7] there is a slight though distinct flattening of the crest of the femoral head, and a slight increase in the distance between the femoral epiphysis and the bottom of the acetabulum. A faint zone of radiotranslucency is found beneath the joint cartilage, due to resorption of necrotic bone, and a slight radiotranslucency at the epiphyseal disc. This latter finding is reported by Howorth and Ferguson,[4] who also report changes in the metaphysis consisting of widening of the cartilage zone, with woolly appearance on the diaphyseal side, and a line of increased density on the epiphyseal side.

In a slightly later stage, there is an increase in the density of the femoral capital epiphysis, as compared to the normal side.[8] The head appears smaller and slightly flattened at the crest[4] and there is a relative increase in the joint space. Osteoporosis of the adjacent extremity of the diaphysis is seen often, which may be manifested as linear zones of translucency running obliquely downward and outward from the metaphysis, or as a zone of translucency of uniform depth across the proximal extremity of the diaphysis, or as one or more circumscribed areas of translucency which may suggest a cyst in the diaphysis near to or abutting on the metaphysis, or away from it in the neck.[8] This stage is often difficult to differentiate from an early tuberculous process, but careful study of the relative densities of the affected and normal sides usually gives the clue to the diagnosis, since in tuberculosis the affected side shows local atrophy instead of increase of density.

The moderately advanced cases present little difficulty in diagnosis. The head is usually markedly flattened and may show evidence of compression fractures.[8] Areas of increased density separated by areas of decreased density are prominent and progress until the head appears fragmented.[4,8] The femoral neck becomes broader and the upper angle rounds off, giving an aparent coxa vara deformity.

The main differential diagnosis in this disorder is from tuberculosis of the hip. As has been mentioned, in the early cases, the only differential point may be the increase in density of the head. Serial films are important in early cases, especially if the diagnosis is doubtful, and if the case is one of osteochondritis deformans juvenilis, the typical deformity will show up in a relatively short time.

Slipping of the upper femoral epiphysis has been a rather rare finding in our cases, but Brailsford[9] reports that the frequency between the ages of 12 and 21 is equal to that of tuberculosis of the hip in this age group. The etiology is not definitely known, and would appear to be variable. Regardless of the primary cause, it is usually agreed that slipping does not occur when the metaphysis is normal, but that some change must take place in it before any displacement occurs. Theories as to etiology may be divided into three groups: disease, static causes, and trauma. Under disease are included acute pyogenic infections. These may destroy the metaphysis, but the usual result is the separation of the epiphysis as a sequestrum. Renal rickets is believed by Brailsford[9] to be the cause in a considerable number of cases. Howorth and Ferguson[4] are of the opinion that circulatory disturbances are the primary factors, as in coxa plana. They maintain that during the years from 9 to 12 the epiphysis is more susceptible or vulnerable to changes in the blood supply than the metaphysis, but that during the years from 9 to 16 the metaphysis is more vulnerable, thus explaining the age incidence. Endocrine disorders are reported by Scott[10] to be present in about 50 per cent of his cases. Static causes have been pretty well ruled out as primary factors, as has trauma. In either case, the consensus of recent opinion is that while they may be the precipitating factors, some change in the metaphysis must have preceded them.

Roentgenologically, the disorder may be divided into the pre-slipping stage, and the acute, or post-slipping stage. In the pre-slipping stage, there is no evidence of erosion or destruction of the bone or joint. The metaphysis is thickened. The diaphyseal border of the metaphysis ap-

pears woolly, while the epiphyseal border shows some increase in density, forming a well defined line.[9] This same change has been reported by Howorth and Ferguson[4] to be found in early cases of osteochondritis deformans juvenilis.

The acute or post-slipping stage is fairly characteristic. The margins of the neck of the femur are higher than the corresponding margins of the epiphysis, the amount of deformity corresponding to the degree of slipping. The epiphysis slips downward and backward, with some inward rotation, giving the appearance of some flattening of the head.[9 11] The metaphyseal periphery of the epiphysis is clearly defined and regular, the shape is that of a cap which has slipped off the upper end of the femur. A distinct gap is noted between the neck of the femur and the epiphysis at the upper end of the epiphyseal line.[11] Pressure of the upper end of the femur against the upper margin of the epiphysis tends to increase the deformity. The neck of the femur may show resorptive changes,[11] and Scott[10] reports that aseptic necrosis of a portion of the head was seen in 5 of 38 cases in his series, the apex usually being involved. Following reduction, there is fairly rapid healing and reconstruction, comparable to traumatic slipped epiphysis. The epiphysis tends to fuse early. In cases not properly reduced, where the head and neck are malunited, there is a distinct increase in the width of the femoral neck, with a fairly pronounced coxa vara deformity.[11] The head in these cases is frequently flattened and somewhat deformed, though not always.

It is doubtful that all cases of epiphyseolysis and osteochondritis deformans juvenilis belong in the same general grouping, but the presence of metaphyseal changes in some of the cases of the latter, and aseptic necrosis of the head of the femur in some of the former would seem to indicate that in a certain percentage there is a definite relationship.

Coxa magna is not usually considered as a separate entity, the roentgenologic findings of a broadened head and neck usually being interpreted as the end result of osteochondritis deformans juvenilis, slipped epiphysis or some other pathologic process. Howorth and Ferguson[12] believe, however, that it may exist as a separate entity, no changes indicative of any other process showing up. In their series of cases, the patients were first examined from within a few days to two years after the onset of symptoms, and followed up until recovery, the only findings being an elongation of the neck and enlargement and broadening of head and neck. The clinical symptoms are those of coxa plana or slipped epiphysis, and the condition may follow subacute arthritis, reduction of congenitally dislocated hip, or a previous suppurating arthritis. All of these conditions may cause sclerotic changes about the femoral neck, embarrassing the circulation. If the circulatory disturbances are great enough, coxa plana results instead of coxa magna. The pathologic changes found on exploration consist of thickening of the synovial membrane and capsule, with vascular engorgement. The vessels are usually thick walled. The cartilage sometimes appears pitted and the bone vascular. The periarticular tissues may appear sclerotic instead of thickened and engorged. These same changes are also found in early coxa plana and the pre-slipping stage of epiphyseolysis. The x-ray findings consist of broadening of the femoral head and neck. The joint space may or may not be widened. The joint capsule may or may not be distended. As the disorder progresses, the broadening of the head and neck of the femur tend to increase for a varying time; the other changes revert to normal.

In the end stages of coxa plana and in epiphyseolysis with faulty reduction, marked broadening of the head and neck also occurs. The deformity may be extremely irregular in shape, or may present a smooth contour, the neck appearing shortened, and the head markedly enlarged and bulging from the socket. Whether or not this process exists as a separate entity we are unable to confirm or deny from our own cases, inasmuch as ours are all the end result of some other process.

Tuberculosis of the joints is an ancient and well known disease entity, having been first described by Wiseman in 1676. The clinical picture is well known, as is the pathologic picture, but for the sake of clarity, the latter will be briefly reviewed.

The disease process is primary either in the bone or in the synovial membrane of the joint. Tuberculosis of the synovial membrane usually begins at the circus vasculosus, the plexus of vessels at the reflexion of the membrane.[13 14] The disease spreads out along the membrane and also spreads over the joint cartilage in the form of a pannus of fibrous tissue, with later a few tubercles. At the same time, there is an invasion of the subchondral region by granula-

tion tissue. There is no tubercle formation in the cartilage, the destruction being by invasion by granulation tissue both from above and below, from the pannus and the subchondral granulations. The pannus formation is along certain routes, the free margins of the cartilage being covered first, and the weight-bearing areas last. The subchondral invasion is one of the earliest changes in the joint, the entire joint cartilage sometimes being undermined before much evidence of disease is noted. The cartilage is destroyed slowly due to the lack of proteolytic ferments in the joint exudate. For this reason, the joint space is often fairly well preserved even in advanced cases. Invasion of the underlying bone from the subchondral granulations may occur, though the first bone changes seen, are usually at the margins in the region of the circus vasculosus. Since this is also the starting point for synovial tuberculosis, the question of which develops first is difficult to determine. The time interval is certainly not great, and the joint and bone invasion may proceed synchronously.[14]

Primary osseous tuberculosis is relatively more frequent in children. Metaphyseal involvement is more common in the younger age groups, with some primary epiphyseal involvement in late childhood and adolescence. Epiphyseal lesions always, and metaphyseal lesions usually, spread to involve the joint. Metaphyseal lesions usually spread to the diaphysis, but may spread to the epiphysis and then into the joint by direct invasion or via the synovial membrane. The bone lesion is usually primary in the epiphysis in the adult.[15]

The pathologic picture varies. There may be only scattered tubercles, each more or less encapsulated with fibrous tissue. Again there is an abundance of soft tuberculous granulation tissue which exerts an erosive action on the trabeculae, causing enlargement of the haversian canal. Branching trabeculae are left, the interspaces being filled with granulation tissue. This process is called caries. At times marked caseation and cavitation occur with occasionally the formation of cold abscess. The process may appear localized, consisting of a caseous center surrounded by a zone of caries with an outer zone of fibrous tissue. In other cases it spreads too rapidly to form caries. Instead, bone necrosis is found and sometimes large sequestra are formed. In addition to the direct effect of the tuberculous process, there is usually a well marked atrophy of the bone.[14 16]

The x-ray diagnosis is often difficult in the early stages. Widening of the joint space is sometimes seen, due to effusion.[13] Soft tissue swelling is seen, and haziness or blurring of the bone structure of the end of the bones. This haziness is attributed to the increased thickening and vascularity of the synovial membrane.[13 14] One of the most significant findings is a localized diminution of lime salts.[13] This one finding is often the only clue to the identity of the disease process at this stage.

In moderately advanced cases, a marginal erosion of the bone may be seen at the peripheral junction of bone and cartilage.[13 14 15] Irregularity or loss of the articular cortex in the non-weight-bearing areas is found. The joint space is preserved as a rule.[15] This is due to the fact that the non-weight-bearing portions are destroyed first, and also the fact that tuberculous pus apparently lacks any proteolytic power.

Advanced cases show a narrowing of the joint space. There is erosion and loss of the entire bony articular cortex, though some joint space may still be present. Kissing sequestra may be formed at the area of closest contact and greatest pressure by secondary bone involvement, though these are not commonly seen in treated cases. These sequestra appear as roughly cone-shaped areas of greater density, and often show preservation of the articular cortex. When the primary focus is in the epiphysis, wedge-shaped sequestra are sometimes seen. In these, the articular cortex is also preserved. In the advanced and moderately advanced stages, extreme atrophy is apt to be a prominent feature.[14 15]

Childhood tuberculosis beginning in the metaphysis presents a different picture. An oval or cone-shaped area of rarefication bordering on the epiphyseal line is seen, which may contain bone and small sequestra. Periosteal and endosteal new bone formation is often seen. The commonest location is the inferior and anterior portion of the femoral neck, the area of rarefication involving almost the entire neck, often with broadening of the neck due to the new bone formation. Involvement of the joint with breaking down of the articular cortex of the head and acetabulum and narrowing of the joint space is common. The joint changes are the same as with primary synovial tuberculosis described above.[15]

Epiphyseal tuberculosis, which usually begins in the region of the circus vasculosus, presents the same picture as synovial tuberculosis, with

perhaps earlier involvement of the bone at the margins.

Childhood and adult hip involvement differ somewhat, due mainly to the increased thickness of the articular cartilage in children. The underlying bony cortex is destroyed more slowly, giving x-ray changes later in the course of the disease. When the bone is secondarily involved in children, there is more apt to be cavity formation in the opposing bone surfaces instead of sequestra.[15]

The chief micro-organisms responsible for acute pyogenic arthritis are the *Streptococcus hemolyticus, Staphylococcus aureus,* or the gonococcus. The pathologic picture is, generally speaking, the same, regardless of the infecting organism. The synovial membrane is probably first involved. The changes produced are those seen in any other tissue with pyogenic infection. Fibrinous exudate is formed early on the surface which later shows beginning organization. Areas of necrotic tissue are found. With each exacerbation, reinfection, or secondary infection, another layer of fibrinous exudate is laid down. The articular cartilage shows a fairly early and rapid distintegration with the more active infections. The cartilage appears to be in a gelatinous state, the edges are largely separated and the entire cartilage is often undermined. Pus cells may be seen in the cartilage itself and the cartilage cells have mostly disappeared. In the more advanced stages, the cartilage may be entirely destroyed, leaving the bone exposed. The destruction is most rapid in the weight-bearing areas. Complete destruction of the cartilage may occur in a relatively few days in a severe pus infection. The exposed bone is rather readily invaded by the infectious process with the production of bone erosion and necrosis. In children, and especially in the hip joint, necrosis of the entire epiphysis may occur with the formation of a large sequestrum. In cases where the primary process is in the bone with later extension to the joint, the bone changes are more extensive.[14]

The x-ray findings in the very early or mild cases are inconclusive. The only changes found consist of soft tissue swelling and evidence of joint effusion.[15] In further advanced or very severe cases, the cartilage is thinned or destroyed, primarily over the weight-bearing areas. Destruction of the articular cortex under the destroyed areas of cartilage is seen. Complete destruction of the cartilage and articular cortex may be seen in very severe cases. Regional disuse atrophy may be seen if the process has been present for sufficient time. The characteristic picture is that of narrowing of the joint space in the weight-bearing portion with destruction of the articular cortex. The articular cortex is preserved in the rest of the joint. The above changes may occur in one to two weeks. The picture is exactly opposite to that seen in tuberculosis. Extensive bone destruction is sometimes seen and osteomyelitis rarely. Death of the entire epiphysis may occur. In cases where the joint is involved secondarily to an osteomyelitis of the bone, the bone shows the greatest amount of destruction and the oldest process.[15]

Degenerative arthritis, also known as hypertrophic and osteo-arthritis, is a fairly common disorder in middle-aged and elderly people, though as a rule the process has progressed past the initial stage when seen by the roentgenologist. The pathologic picture is well known. The earliest change seen is a fibrillar degeneration of the joint cartilage in the weight-bearing areas. This process progresses with gradual wearing down of the cartilage until it is very thin or disappears entirely. A compensatory hypertrophy may take place on the opposing articular surface. Calcification may take place in the deeper layers of the cartilage. The articular cortex of the bone becomes thickened by the laying down of new bone, which merges with the calcification in the cartilage. The greatest thickness is in the areas where the cartilage is thinnest or absent, and forms an area of eburnated bone. Proliferation of cartilage and new bone formation occur at the periphery, producing exostoses and increasing the circumference of the head. Beneath the layer of eburnated bone, the bone may be atrophied. There takes place a fibrosis in the narrow spaces, and the spaces may be so enlarged as to give the appearance of cystic degeneration. The synovial membrane may show more or less proliferation of the villi.[14]

On x-ray examination, soft tissue swelling is sometimes seen, but not usually. Irregularity of the articulating surface is seen, with some narrowing of the joint space over the weight-bearing areas especially. A slight amount of lipping is apt to be seen at the margins of the joint, and small cyst-like cavities in the juxta-articular bone, especially in the superior lip of the acetabulum and the approximated surface of the femur, may be the earliest signs of arthritis. If the condition has been present for

some time, some thickening and irregularity of the zone of provisional calcification may be seen. Bone atrophy may or may not be present, depending on the amount of disability the patient has experienced. In the advanced cases, the typical picture of marked thinning of the joint space, sclerosis of the weight-bearing surfaces of the bones, and exostoses is seen.[8 14 17]

Cases of Paget's disease and carcinoma with metastasis to bone are sent to the roentgenologist often for pain in or around the joints. We will not attempt to discuss the pathologic picture or x-ray characteristics of these conditions because neither is strictly a joint lesion. We do wish to point out, however, that in neither condition, regardless of how advanced the changes in the bones may be, do changes in the joint itself occur. In practically every case, the joint space and cartilage are preserved, although changes may be seen in the bones, running up to and involving the bony articular cortex.

CONCLUSION

After reviewing our cases and the literature, one thing perhaps stands out most clearly, and that is that with the exception of congenital dislocation of the hip, and possibly slipping of the upper femoral epiphysis, the earliest roentgenologic findings of hip disorders are not diagnostic. This emphasizes the need for serial examinations, and in any case presenting the picture of soft tissue swelling, haziness of detail, preservation of the joint space and localized bone atrophy. These should never be neglected. Tuberculosis cannot be positively diagnosed on these findings, but it should always be strongly considered as present until ruled out by later examinations.

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DISCUSSION (*Abstract*)

Dr. Ernst Lachmann, Oklahoma City, Okla.—Roentgenology has come of age and so it is worth while from time to time to take stock on what we can diagnose and what we cannot demonstrate with x-rays. In this respect I agree especially with the guarded conclusions expressed by Dr. Johnson. His statements that the early x-ray findings in lesions of the hip joint are not pathognomonic are very much to the point.

Radiologists have always been very anxious to check up on their interpretations whenever an opportunity arose, at autopsy or operation. Unfortunately it is often only the advanced lesion which allows such a comparative study. The animal experiment to which we are accustomed in other branches of medical science fails us here because proportions and dimensions are different from what we find in man.

As a radiologist who had the good fortune to work in an anatomical laboratory with an ample supply of bones, I approached the problem of early x-ray signs of destructive lesions from a different angle. Small defects of varying size and location were produced in bones and the bones x-rayed in standard positions. The defects were filled with paraffin or paraffin and bone detritus to imitate bone disease. Control pictures of the untreated bones and of the defects filled with opaque material were taken. The size of the defects was determined by measurement and demonstrated by filling with opaque material. We usually started with small excavations which were gradually enlarged. This gave us an opportunity to recognize the faintest signs of destruction and to mark out the limitations of roentgenologic procedure.

Our results show that not all osseous defects are visible on the roentgenogram in either frontal or profile view, but that they require a certain size in order to appear on the x-ray film. The minimum dimension necessary for visibility varies with the location of the defect. Conoid shaped excavations involving only the spongious structure require a diameter of from 0.5 to 1.9 cm. Disc-like cortical defects must have a diameter of from 0.5 to 2 cm. in order to be seen. Some locations are especially favorable; others are especially unfavorable for visibility of destructive foci. Different projections are very important for their demonstrability. Factors determining visibility of a defect are: (a) direction of its longest axis in relation to the central x-ray beam; (b) diameter of transradiated bone which is superimposed over the defect; (c) relative amounts of cortical and spongious matter in the overlying bone; (d) distance of the defect from tube and film; (e) character of border excavation; (f) content of defect; (g) state of calcification of the surrounding bone.

In the light of these experiments the limitations of

x-ray procedure in the diagnosis of early destructive lesions gains new significance.

Dr. Johnson (*closing*).—Dr. Lachmann's discussion is slightly outside the intentions of this paper on hip joint disease. Of course, as he has demonstrated, in long bones and about the knee joint, certain types of fair sized areas of destruction may be produced without showing on the x-ray negative and he is to be congratulated on the immense amount of work done to bring out this point. In the hip joint, however, we have the ligament, weight-bearing, and so on, to contend with, but even in this area the disease is generally much more extensive than the actual showing on the x-ray negative.

Two points in our paper we consider exceedingly important. One of them is the slight amount of change that may be present in virulent streptococcic joint disease, with the rapid recovery and slight permanent damage after the sulfanilamide is given. The other one is the early increased calcification in Perthes' disease compared to the early decreased calcification or decalcification of incipient tuberculosis.

UROLOGIC DETERMINATION OF FECUNDITY IN THE MALE*

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Contrary to the opinions of many of our younger medical men, there is a greater demand for scientific information concerning sterility than there is for contraceptive data, the latter being dispensed freely by well-meaning, but often misinformed, friends and relatives.

The responsibility for nonproductive marriages is now recognized to be of dual origin. The role of the male has undergone a change from a state of complete innocence to one of contributing guilt in a high percentage of cases and to complete guilt in a noteworthy number.

Within the last few years the study has progressed to a more scientific and accurate basis on a foundation of actual case histories. Haphazard analyses have been found of very little value. We are beginning to realize that, with a little effort, thoroughly informative examinations can be done in the average office laboratory.

*Read in Section on Urology, Southern Medical Association, Thirty-Second Annual Meeting, Oklahoma City, Oklahoma, November 15-18, 1938.

It was noted in the Nineteenth Century that spermatozoa rapidly died in a highly acid vagina, but it was not until 1913 that Huhner[1] stressed the importance of male responsibility in the sterile marriage. His idea of aspirating the testes and epididymis when the ejaculate was repeatedly found to be without spermatozoa still is thought a valuable test. Hagner,[2] however, deprecates the use of puncture because of possible injury to the testicle.

In 1914, Cary[3] advocated examination of the male before subjecting his mate to gynecologic examination or treatment. At that time he believed 35 per cent of sterile marriages to be caused by seminal defects.

Macomber[4] proposed the hypothesis of relative fecundity in an individual with the theoretical perfect being 100 per cent. Perfection is rarely seen in either male or female. The relative fertility of both parties must be high. A male and female, both potentially fertile but with low relative fecundity, may have a fruitless marriage. Either of these, if crossed with a partner of high relative fecundity, may have a fruitful marriage.

In undertaking the analysis of an individual from a standpoint of potential fecundity, an accurate and detailed history is, of course, of inestimable value. In order that no important statement be lost, we use a history form especially applicable to such patients. With the exception of but one or two minor changes, it does not differ in any respect from that suggested by Meaker and his co-workers at Boston. It includes such variable and possibly contributory points as the following:

(1) Present marriage, contraceptions, pregnancies.

(2) Previous marriages, with the same subheads.

(3) Sex Life: ignorance of sex technic; extent of unphysiologic intercourse.

(4) Venereal disease: its effect upon all organs of procreation.

(5) Past injuries, including x-ray exposure and mumps.

(6) Other urologic diseases, operations and hernias.

(7) Present urologic symptoms.

(8) Previous sterility examinations and treatment.

The multiple marriages of modern life have

assisted in our study, broadening the scope of our field of observation, by affording in some instances an opportunity to compare the present marriage status with the former one.

The physical examination should begin with a close inspection of the general appearance of the individual. This is best done with the patient nude so that it can be determined whether there is any variation from the normal in his male development. The well-known sex characteristics should be carefully studied with particular attention to the question of glandular deficiency.

A careful inspection of the genitals is next in order. Underdevelopment is frequently seen, and can be corrected only before puberty, for afterward the response to therapy becomes unfavorable. It should be the duty, therefore, of every physician examining male children to keep this thought in mind.

Phimosis, hypospadias and epispadias are of definite clinical significance, for these defects may often result in the deposit of spermatozoa in an unfavorable location. Scars are suggestive of an old primary lesion. The size of the meatus and urethra should be determined and boule' a boule' used to examine for strictures. Hernia and varicocele often interfere with circulation to the testes.

Undescended testes rarely, if ever, produce spermatozoa. The size, shape and consistency of the testes are important, for hypoplasia is often associated with oligospermia. The epididymis must be examined for evidence of old inflammatory changes or tumors. A smear should be taken from the urethra, followed by the two-glass test, and an examination of the prostate gland and seminal vesicles. Particular consideration is placed on pus and bacteria from the prostate and vesicles, and the secretion is examined for spermatozoa.

Examination of the ejaculate should be made following 3 to 5 days' sexual rest. Collection of the specimen is of prime importance and is most satisfactorily accomplished by using a wide-mouth glass container, such as a small mayonnaise jar or jelly glass. No particular effort need be made to maintain a fixed temperature, inasmuch as the spermatozoa have been found to withstand considerable variation. Kingman restored motility to bull spermatozoa that had been frozen. A clean glass container is far superior to collection in a rubber condom, inasmuch as contact of semen with rubber depresses motility and frequently results in a complete necrospermia, even when the condom has been carefully washed and dried. The specimen should be examined as soon as possible, preferably within thirty minutes of ejaculation.

An accepted analysis (Vose, Meaker, Hotchkiss) of the fresh specimen should contain the following:

(1) The physical appearance and viscosity. The normal ejaculate is gray in color and is viscous if seen immediately. The viscosity disappears in from one-half to one hour due to a mucolytic action, which facilitates the motility of the spermatozoa. The same principle liquefies the thick, mucous plug of the cervix, and allows spermatozoa to reach the uterus (Kurzroh *et al*).

(2) The hydrogen ion concentration of the specimen should be determined. It normally ranges from 7.7 to 8.5. It is thought by some authorities (Spicer) that the spermatozoa travel toward, or are attracted by the higher pH in the cervical mucus. Meaker and Glasser have shown that the cervical mucus is constantly alkaline and that in 80 per cent the pH is 8.5 or above. Litmus or nitrazene papers are usually satisfactory for this determination; however, other indicators are more accurate. High acidity of the vagina has been repeatedly demonstrated as detrimental to spermatozoa.

(3) The average amount of semen varies from 3.5 to 6 c. c. Less than 3 c. c. is thought to be inadequate in the average union; however, examination of one specimen is not sufficient to determine this average quantity. Small amounts of semen may be caused by a reflux into the bladder due to obstruction produced by any congenital anomaly or stricture distal to the ejaculatory duct. In all cases of oligospermia the first urine following coitus should be centrifuged and examined for spermatozoa.

(4) The average number of spermatozoa is 100 to 150 million per c. c. The total ejaculate, therefore, should contain 350 to 500 mil. Amounts less than 60 mil per c. c. are less likely to cause pregnancy. Recently, sulfanilamide has been shown to depress spermatogenesis (Barbellion and Tarres-Leon, Siecle Medical 7-1-38).

Dilution in the hemocytometer is misleading and unsatisfactory. One c. c. should be collected in small amounts from various parts of the semen[6] and diluted from 1-20 to 1-80 in a solution of 1 per cent formalin and 5 per cent sodium bicarbonate as has been proposed by Macomber. The count is made on any standard

blood counting chamber. Spermatozoa are not uniformly distributed in the diluted specimen; therefore, several drops should be examined, as there is variation up to 20 per cent from the mean.

(5) Motility is difficult to describe and the normal motion is learned by repeated observations of specimens. There is a whip-like action of both head and tail and frequently an associated rotary motion. In normal specimens, 80 to 90 per cent are motile. Veterinarians recognize the second "service" of "fresh" semen to be more motile than the first. Abnormal forms are frequently as motile as the normal.

(6) Moench and co-workers describe a technic of intricate measurement of spermatozoa, which is definitely more accurate than the usual method, yet requires considerable time and equipment that is not a part of the average office laboratory.

The fresh unstained specimen is sufficient to determine the presence of many types of abnormal forms, but a stained specimen is necessary to make a percentage count of these abnormalities. The stained specimen also reveals the presence of some abnormal forms that are not recognized in the fresh specimen.

Various solvents and staining technics are used. It is well to mix one drop of semen and one drop of blood serum, then fix by heat. This prevents the preparation from washing off the slide. The galea or tip of the head takes a lighter stain than the base of the head. In our practice we have used with success the staining method of Williams described by Vose.[6]

Among the abnormal types are long head and body, double heads and tails, solidly staining small heads, large heads, and heads with frayed surface membranes. Immature forms are frequently encountered with a large part of their original cytoplasms still attached. Many forms with acute angulation of the body may be found.

(7) Endurance of motility lasts from eight to twenty-four hours under sealed cover glass at room temperature. Warm specimens are more motile, but have less endurance; cold specimens are less active but have more endurance.

May we digress here in order to mention briefly two cases which are illustrative of some of the factors which we have stressed.

Case 1.—A white male, aged 29, a clerk, has been married 6½ years and his wife had not been pregnant. Sex life was normal as to duration, frequency and reaction. The past history was entirely negative. The parents had seven children, and one of the brothers had two healthy children. The patient had had no previous fertility examination, but his wife had been subjected to numerous pelvic examinations and a tubal patency test.

Examination of the male revealed a well-nourished, healthy individual with scant distribution of hair on his face. There was no hair on the chest or abdomen, and only a small amount on the mons pubis. The patient's voice was normal. Observation revealed hips of feminine contour. The penis was short and had a connective tissue band on the ventral surface like that seen on hypospadiacs. The meatus had small lateral labia, but was normal in size and location. Folds of skin from the scrotum continued upward on each side of the penis to converge on the pubis. Examination of this patient's ejaculate revealed the amount to be 4.2 c. c. with a viscosity and color normal; pH 7.5 plus; but no spermatozoa could be found. Unfortunately, this patient moved to another city, and no further study has been made during this time to our knowledge. Had a simple fecundity examination been made first on the husband, the extensive examinations on his wife would have been unnecessary.

Case 2.—A white male, aged 38, had been married eight years. His wife became pregnant one year after marriage, and the result was a monstrosity. She became pregnant again several years later and had a spontaneous abortion after two months. The patient's sex life was normal and no contraceptive measures had been taken during the previous three years. Pre-union analysis of semen several years ago showed many abnormal forms. Rubin test and pelvic examinations recently made on the patient's wife had all been normal.

Physical examination was essentially negative. The semen analysis was as follows:

Quantity, 6 c. c.

Viscosity and color, normal.

pH, 7.5 plus.

Count, 80 million per c. c.

Motility estimation, 70 per cent.

Endurance good, with twelve hours' activity.

Morphology showed 30 per cent abnormal forms with some spermatozoa containing two heads and others two tails.

There was no definite conclusion to be drawn from this case, but it does indicate relative sterility, and further suggests that interesting data may be compiled in many of the larger clinics, where monstrosities are not so rare as in our practice.

In closing, may we emphasize the fact that the male more frequently contributes to the cause of barren marriages than has been generally recognized until more recent years. Thus we suggest an examination of the male before subjecting his mate to extensive investigation, since the factors of deficient or abnormal spermatogenesis, obstruction of the conveying channels, infections in the prostate gland and seminal vesicles play an important role, as do various

other factors which may be brought to light after careful examination by the internist, endocrinologist or other special consultant.

While the subject of treatment is beyond the scope of this discussion, it cannot be denied that in a very large percentage of these cases neither operative or nonoperative measures offer any real hope of success.

In all probability, however, no other urologic problem requires more tact, finesse and consideration of the human element than that of dealing with such cases. Many men might feel themselves distinctly inadequate and consign themselves to the life of a confirmed neurotic if suddenly confronted with the fact that they were hopelessly sterile. Either party might look with accusing disfavor upon the other if the responsibility and dissatisfaction were laid too heavily upon one doorstep. In such cases we believe that it is pardonable to keep in mind, and even stress, the undeniable fact that 100 per cent perfection in either male or female is a very rare occurrence. Here, as in every other phase of our work, the cool, clear light of science must be tempered with the softer glow of human understanding.

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DISCUSSION (*Abstract*)

Dr. Edward White, Dallas, Tex.—I wish to discuss this very interesting paper from two angles: first, that of infection in the prostate and vesicles. All of us have seen patients whose semen was loaded with pus and dead sperm, who received routine treatment and whose wives subsequently became pregnant. Whether the infection actually kills the sperm as they enter the vesicles, as they come in contact with the infected prostatic fluid or retards their formation in the testicle is unknown. Possibly some light may be thrown on this by the work being done by various workers in catheterizing the ejaculatory ducts.

The other angle from which I want to discuss this paper is that of the ductless glands. We are all familiar with the fact that in a childless home if a baby is adopted it is not uncommon for the wife to become pregnant within a few years. It has been claimed that this is due to the stimulation of a ductless gland in the foster mother, and who can say that it is not due to the stimulation of a gland in the foster father?

An enlightening article by Norris J. Heckle, of Chicago, appeared in *Endocrinology* of January, 1938, describing the work done on a series of men whose marriages had been sterile for two to five years. Anterior pituitary-like sex hormone was used in enormous doses and although I have had no success with these drugs and had given them up, I intend to retry them, as my efforts were rather crude as compared with this work. Heckle gave large doses daily and checked the sperm count at intervals. He showed that in some cases the count was actually decreased and then increased after the injections were stopped. In most cases the number was increased along with the motility. He was unable to show any improvement in testicles atrophic from mumps.

THE TREATMENT OF ACUTE CHOLECYSTITIS: IMMEDIATE OR DELAYED SURGERY*

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No comparison can be drawn between the treatment of the acute gallbladder and the acute appendix. Such an analogy indicates a lack of basic knowledge of the two appendages. The appendix is an atrophic structure while the gallbladder emerges as a continuous tissue growth to the adult stage. The appendix is a low lying structure contrasted to the gallbladder situated high in the abdomen, in close proximity to the liver, closely allied to the common duct, surrounded by a number of vital organs and sharing in the higher mortality of upper abdominal surgery, due to pulmonary complications. The appendix has no known physiologic function; the gallbladder definitely has. In inflammation, the appendix is infected by a number of virulent organisms whereas the gallbladder undergoes in most instances a circulatory change due to obstruction of the venous return, and organisms are not often cultured even in cases

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of empyema. The gallbladder stroma is marked by a rich vascular anastomosis and the lymphatic supply of the gallbladder and the liver are very closely interrelated.

Three hundred and twenty-two cases of gallbladder disease which had been operated upon were studied in order of their admission from the files of St. Anthony and University Hospitals. Eighty-one cases of acute cholecystitis occurred, the diagnosis corroborated by immediate operation or surgery in the quiescent phase. Approximately one-half of these cases occurred in the fourth and fifth decades of life.

Chart 1

Age	Number	Mortality
10-20	1	0
20-30	5	1
30-40	18	2
40-50	22	0
50-60	22	2
60-70	11	1
70-80	2	0
Total	81	6
Mortality	%	7.4

Forty patients (50 per cent) were operated upon within three days of admission; thirty-seven were operated upon within 24 hours of admission. However, thirteen of these thirty-seven patients (35 per cent) had had symptoms seven days or longer before being hospitalized. Too many gallbladder patients are not hospitalized early. This is probably one of the reasons why the conservative treatment has had much criticism. There is a dividing line between conservatism and neglect.

Chart 2

SURGERY IN 24 HOURS: DURATION OF SYMPTOMS

Days' duration	1	2	3	4	5	6	7	11	21	30
Number	6	5	5	5	2	1	7	1	1	4

Likewise, of thirty-two cases of empyema, nineteen (60 per cent) had had symptoms seven days or longer before admission to the hospital and one patient had had a mass in the right upper quadrant for six months with several attacks in the interim.

Chart 3

DURATION OF SYMPTOMS BEFORE ADMISSION TO THE HOSPITAL, WITH OPERATIVE FINDINGS OF EMPYEMA

Days' duration	1	2	3	4	5	6	7	8-21	Over 21
Number	4	1	3	3	2		4	4	11

Furthermore the acute gangrenous group demonstrated that in spite of the fulminating symptoms that were present, seven of the ten cases (70 per cent) had had symptoms three days or longer before admission.

Chart 4

DURATION OF SYMPTOMS BEFORE ADMISSION TO THE HOSPITAL, WITH OPERATIVE FINDINGS OF GANGRENE

Days' duration	1	2	3	4	5	6	7	Longer than 7
Number	2	1	1	2	2		1	1

Doubtless this delay in hospitalization of the gallbladder patient has a definite bearing on the hospital mortality. Certainly any gallbladder attack that remains unabated at the end of twenty-four to forty-eight hours or subsides in that time to recur, should be hospitalized for close observation, such as is usually possible only in an institution.

Not too much reliance can be placed on the degree of leukocytosis and the differential count, unless it is excessively high or in certain cases unusually low. In cases operated upon within twenty-four hours of admission there was a marked variation in the white count regardless of the duration of symptoms. In most instances, however, there was a definite leukocytosis.

As a general rule, the more severe lesions such as the gangrenous gallbladder produce the higher count and this is helpful in making the decision for prompt surgery. Six of the ten gangrenous gallbladders had a total count over 18,000 and half the polymorphonuclear counts were 90 per cent and over; the temperature was found to be a less reliable guide as to the severity of the lesion. In this gangrenous group the

Chart 5

DURATION OF SYMPTOMS IN RELATION TO THE VARIATION IN LEUKOCYTOSIS

Duration	24 Hours	48 Hours	72 Hours	4 Days
High and low white count	4,400-35,800	8,200-22,200	6,500-17,600	6,200-18,200

Chart 6

TEMPERATURE, WHITE COUNT, DIFFERENTIAL AND DURATION OF SYMPTOMS BEFORE OPERATION IN GANGRENOUS GALLBLADDERS

T.	WBC	Dif.	Duration Days	Result
98	22,800	96	1	Well
101	20,300	93	2	Well
102	22,000	95	4	Refused surg. 2 da. exp.
101	10,500	84	4	Well
99	21,650	89	5	Well
99	18,200	91	5	Well
100	23,700	84	5	Well
101	12,500	90	7	Well
98	9,200	84	7	Well
	10,500	72	7+	Well

Chart 7

THE WHITE COUNT AND DIFFERENTIAL ARE OF LESS VALUE IN CASES OF EMPYEMA AS SHOWN IN THIS TABLE

WBC	Number	Duration of Symptoms
4-6000	1	7 da.
6-8000	6	Chr. 7d. 7d. 3d. 3wk. 3d.
8-10,000	5	Chr. chr. chr. 5d. 6mo.
10-12,000	8	5d. 3wk. 1d. 11d. 2d. 4d. 1d.
12-14,000	3	3m. chr. 4d.
14-16,000	4	4d. 10d. 14d. chr.
16-18,000	2	3d. 1d.
18-20,000	1	1d.
20,000 up	2	2d. 4wk symptoms, violent pain 24 hours, rupture

Temperature Degree	Number	Differential and Number	
97	1	50-60	1
98-99	5	60-70	2
99-100	5	70-80	9
100-101	8	80-90	12
101-102	4	90 up	8
102-103	7		
103 up	2		

longer the duration of symptoms the lower the total count.

The empyema cases showed a greater variation of the temperature, white blood count and the differential. However, as has been indicated there was more contrast in the duration of symptoms.

In the three perforated gallbladders with general peritoneal contamination, the counts were respectively 25,000, 35,700 and 6,500. These were helpful, for because of the clinical findings they were indicative of a fulminating process, accompanied on one hand by a normal count and on the other by a marked elevation.

That a gallbladder can be in an acute stage and be characterized by relatively mild clinical findings has been conceded by several authors. Six of the three hundred and twenty-two cases operated upon presented acute findings at surgery. They had little indication that such a condition was present, other than an elevation of the temperature, white blood count or differential. An empyema was found in four and acute inflammation in two of these cases.

Fourteen of the eighty-one acute cases (17 per cent) had no history of previous attacks. These included two empyemas, one pericholecystic abscess, one gangrenous and one ruptured gallbladder. Two of these cases were 70 years or over. Surgery within twenty-four hours was necessary in six.

Recent extensive changes in the gallbladder wall are usually associated with obstruction of the cystic duct. Such obstruction is produced most often by an impacted stone and less frequently by inflammatory edema. A circulatory change occurs in the wall, dependent on the degree of obstruction. This varies from edema

Chart 8

CASES TO SURGERY WITH MILD OR NO SYMPTOMS AND PRESENTING ACUTE FINDINGS

Sex	Age	T.	WBC	Diff.	Procedure	Findings, Result, Hospital Days	
F	32	100.2	12,350	86	Removal	Thick wall, stones, Ac. Inf., adhesions Imp. large gallbladder	18
M	49	99.8	12,400		Removal	Thick wall, acute inf., adhesions, pus exp. uremia	6
F	55	98.8	6,700	68	Removal	Thick wall, necrotic Imp.	34
M	59	99	9,800	80	Removal	Thick wall, stones, pus, ac. inf., adhes., exp. pulm. emb.	18
F	35	100.6	10,200	80	Removal	Thick wall, stones, pus, adhesions, large gallbladder, exp. pulm. emb.	11
F	49	98	9,200	88	Removal	Thick wall, stones, pus, adhesions. Imp.	20

Chart 9

Duration of Attack, Days	Age	Sex	Temp.	WBC	Polynuclear Per Cent	Interval to Surg. and Proc.	Findings	Result	Hospital Days
7	35	F.	97	10,000	97	24 h., inc. and dr.	Stones	Well	19
8	72	F.	101	20,950	77	5 d., removal	Small, thick wall, adhesions	Well	21
2	70	M.	100.6	10,050	90	Immed. removal	Acute, stones, pus	Well	21
3	48	F.	102	7,450	92	7 da., removal	Pus	Well	29
7	28	F.	99	14,250	80	5 da., removal	Stones	Well	19
7	53	F.	98.6	7,300	63	1 da., removal	Small, thick wall, adhesions, stone	Well	?
24	61	M.	99.4	5,900	60	3 da., inc. and dr.	Acute, stones	Well	25
4	53	M.	99	18,200	91	24 h., inc. and dr.	Gangrenous, stone, adhesions, thick wall	Well	25
21	40	M.	101	10,000	92	5 da., inc. and dr.	Pericholecystic abscess, stone, adhesions	Well	50
1	33	M.	99.4	4,300	70	4 da., inc. and dr.	Adhesions	Well	47
13	30	F.	99.4	6,800	56	3 da., inc. and dr.	Thick wall, adhesions	Well	21
5	45	F.	101.2	27,000	90	Immed. removal	Thick wall, stone, acute, ruptured	Well	19
7	17	M.	103	21,300	90	Immed. inc. and dr.	Acute, distended thick wall	Well	41
9	24	F.	99.8	13,200	92	Cons. 1 mo. Inc. and dr.	Stones, adhesions	Well	37

and venous distention to intramural hemorrhage as in an infarction. Perforation of the gallbladder follows stone erosion of this infarct, and sometimes very early in the course of the disease.

Ordinarily it would be inadvisable to open the common duct in the presence of an acute gallbladder. As a rule it is inaccessible because of adhesions, edema, and distortion of landmarks. All structures are involved by the inflammation, and dissection produces hemorrhage and hampers the surgeon who is already working against time. Nevertheless, if routine surgery were performed for the acute gallbladder one would be faced with the possibility of a common duct stone. This is true for two reasons: First, Lahey[1] found that 37 per cent of the patients with common duct stones had no history of jaundice at any time and were not jaundiced at surgery, and second, since most acute gallbladders contain stones (*Chart 10*), about one of every five common ducts would have stones. Furthermore the indications for opening of the common duct, such as palpation of the stone, thickening of the pancreas or the duct, dilatation of the duct, valuable leads as they are in the chronic case, would be useless in the presence of acute gallbladder disease. Lahey's findings were corroborated by Crump[2] who in routine autopsy material of one thousand subjects found three hundred twenty-five gallbladders with stones and seventy-eight common ducts containing stones. In other words, 24 per cent of the gallbladder subjects had stones in the common ducts.

Chart 10

STONES FOUND IN ACUTE GALLBLADDERS

Author	No. Cases	Cases with Stones	Per Ct.
Judd,[3] E. Starr	508	484	95
Miller,[4] R. H.	200	160	80
Clymer, C. E.	81	67	83

In an analysis of the literature it is evident that the acute gallbladder in no small way contributes to the higher mortality brackets. Heuer[5] collected reports of 36,623 gallbladder operations from the literature with 2,453 deaths or a mortality of 6.6 per cent and 1,274 cases of acute

cholecystitis with operations and a mortality of 111 cases or 8.7 per cent. Hotz[6] in a paper presented to the German Surgical Association in 1923, produced figures demonstrating that the mortality of operation during an acute attack was double that which followed operation after remission. Lipshutz,[8] Stone, Zinninger and others have published articles recently favoring the prompt surgical attack.[9] Yet many prefer the conservative view.

Among cases from the St. Anthony and University hospitals, three were perforated and two had pericholecystic abscess. Of the three frank perforations, two patients who had bile in the peritoneal cavity recovered, and one who had frank peritonitis died. The two patients with pericholecystic abscess recovered. Perforation of the gallbladder has a high mortality.

Boyce,[10] analyzing 26 deaths from empyema, gangrene, and rupture of the gallbladder, found that only five were patients whose operation had been delayed in the hospital, whereas, twenty-one had delayed seeking medical treatment until they had reached this state, the operation being done as an emergency measure as soon as they were admitted. In this series of cases, all the mortality of the acute suppurative group occurred in patients operated upon after the fifth day of symptoms. The duration of symptoms before admission in patients who died was, respectively: 4 weeks, frequent attacks; 5 days, frequent attacks; and in the gangrenous gallbladder death, the patient refused surgery for four days. Of the thirty-two cases of empyema, eleven patients delayed hospital admission more than three weeks, one as long as six months, and eight patients delayed seven days to three weeks. In other words, 19 to 32 cases of empyema were hospitalized seven days or more after the onset of the attack. Indeed the total mortality of this series occurred in cases hospitalized five days or more after the onset of the acute attack. Further analyzing this mortality, three died of embolus, one had a ruptured gallbladder on admission, the remaining two died of uremia.

Clinical manifestations indicate the opportune time for operative intervention. In the presence of an acutely inflamed gallbladder, the field is obscured by adhesions, edema and distortion of landmarks. Routine early surgery would increase the mortality and subject more patients to the probability of a second operation because a higher percentage of gallbladders would be drained. Of the thirty-seven cases operated upon in twenty-four hours at our hospitals, cholecystostomy was the procedure of choice in seventeen (46 per cent). In addition to this higher percentage of drained gallbladders, more stones would be missed in the common duct and if cholecystectomies were attempted more common duct injuries would be seen.

If timely intervention is the rule, the patient's best interests are served. Should it be deemed necessary to open the abdomen because of the fear of developing complications, certainly the patient's general condition should be the index to the type of surgery. Granted a patient in good general condition, if too much dissection is not necessary, cholecystectomy is the operation of choice. However, the surgeon should not feel thwarted if only cholecystostomy is feasible. The gallbladder might be a very helpful landmark

Chart 11

MORTALITY STATISTICS ON PERFORATION

Author	Cases	Deaths	Mortality Per Cent
Siegmund: Deutsch. A. F. Chir., 230:359, 1924	8	5	62.5
Danzis: Surg. Clin. of N. America, 6:1397, 1926	1	1	100
Santee: Ann. Surg., 93:1156, 1931	5	3	60
McWilliams: Ann. Surg., 55:235, 1912	6	2	33.3
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Gosset, Deplas: Jour. de Chir., 25:259, 1925	111	?	52.2
Darner, Cullen: Surg., Gynec. & Obst., 37:579, 1923	3	2	66.6
Heuer: Ann. Surg., 99:881, 1934	21		37.7
Clymer	3	1	33.3

should a common duct obstruction subsequently develop. A small crucial incision for observation and to determine the type of operation may be in the patient's favor if a cholecystostomy alone is possible. In the face of a severe pancreatitis, probably the gallbladder should be removed and the common duct drained.

If the attack persists longer than forty-eight hours, the attendant should consider the possibility of an impending complication. Developing gangrene and empyema may become manifest at any time, but more often within 3-5 days of the initial onset. In the presence of fulminating symptoms at any time, or a definite clinical change, one should not hesitate to open the abdomen promptly. Frequent blood counts are helpful and an increasing leukocytosis is important. Meanwhile the patient should be placed on a strict regimen of nothing by mouth, adequate fluids so that the output ranges between 1200 and 1500 c.c. per twenty-four hours, hot packs to the abdomen and not too much morphine.

That complications of gallbladder disease are responsible for most of the mortality in biliary surgery is an established fact. The lowering of this mortality will be accomplished if the physician keeps in mind the high incidence of complications in the hospitalized gallbladders and is more prompt and emphatic in urging early hospitalization to the patient. Allow me to repeat that all the deaths in this series of 81 cases occurred in patients hospitalized five days or more after the onset of symptoms. This to my mind is the crux of the situation rather than routine emergency and perhaps ill advised surgery in acute gallbladder disease.

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PILONIDAL CYST*

By JOHN POWERS WOLFF, M.D., F.A.C.S.
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The fact that many of these lesions have been operated upon and have recurred has prompted a review of the literature and summary of the cases available.

The lesions vary from a mere dimpling of the skin to a sinus extending down to and including the hiatus of the coccyx, which may communicate with the neural canal. Several cases of meningitis have been reported, caused by direct communication of the neural canal with an infected pilonidal cyst.

There are various opinions as to the development of this condition. Mallory[1] and many others believe it to be a remnant of the medullary canal. The possible persistance of the avian preen gland has been suggested by Stone.[2] This is the gland from which a bird obtains oil with its beak to preen its feathers. The consensus of opinion is that the condition is congenital and that the contents are of ectodermal origin. They nearly always contain hair.

True dermoid cysts that contain teeth, bone and cartilage do not seem to be found in this lesion and the pilonidal cyst is not a true dermoid cyst. An occasional lesion anterior to the sacrum is believed to have another origin.

The lesion, as presented to the surgeon, is much more common in the male. The females in our series were all of the type with excessive hair about the region of the sacrum.

The statement has been made that dimpling occurs more frequently in the male. We were unable to confirm this. The surgeon sees lesions more frequently in the male. The reason is obvious, the abundance of hair in this region in the male. The surgeon usually sees only those that have developed infection, and infection is more prone to develop in the hirsuit type of individual. There seems to be general agreement that these cysts, or sinuses, are ectodermal cells growing within this area. The disagreement is only as to the origin, which varies from invagination of the skin to a persistence of a portion of the medullary canal.

A careful block dissection down to the sacrum and tip of the coccyx or including the coccyx, as

*Read in General Clinical Session, Southern Medical Association, Thirty-Second Annual Meeting, Oklahoma City, Oklahoma, November 15-18, 1938.

CONSECUTIVE CASES FROM UNIVERSITY HOSPITAL*

Case	Age	Sex	Symptoms Duration	History	Treatment	Results
1	16	WF		Several previous abscesses followed by convulsive seizures	Abscess, 5 inches in diameter, opened and packed	Results good. Recent communication
2	23	WM	3 yrs.	Incised several times over period of three years	Block dissection closed	Immediate result good
3	18	WM	4 yrs.	Swelling, four years. Tender, painful with drainage past four to six mos.	Excised tract?	?
4	27	WM	1 yr.	Intermittent pain followed by discharge	Dissected, closed. Closure broke down postoperatively	?
5	26	BF	1 mo.	Incised twice	Two small sinuses dissected out, packed and drained	?
6	31	WM	4 to 5 yrs.	Tender, swelling. Four to five years	Block dissection. Closed. Small drain	Immediate result good
7	36	WM	12 yrs.	Ten to twelve recurrent abscesses	Block dissection 30x15x16 cm. closed	Immediate result good
8	23	WM	3 yrs.	Abscess incised three years ago, followed by several recurrences	Block dissection with lateral flap graft	Immediate result good
9	34	WM	7 yrs.	Clear drainage seven years, then painful with intermittent abscess	Block dissection with lateral flap graft	Results good. Recent communication
10	33	WM	2 mos.	Tender with abscess one month. Tender since	Block dissection. Closed with drainage	Results good. Recent communication
11	44	WM	8 yrs.	Incised acute abscess eight years ago. Purulent drainage since	Excision of cyst to coccyx. Left open	Immediate results good
12	3 mos.	WF		Hydrocephalus. Spina bifida. Pilonidal cyst?	No treatment	No treatment
13	24	WM	1 yr.	Noticed small tumor with slight discomfort. Incised twice. Two weeks before became acute	Block dissection. Lateral sliding graft	Immediate results good
14	24	WM	1½ yrs.	Abscess 1½ years ago. Several recurrences	P'ck dissection. Lateral sliding graft	Immediate results good
15	28	WM	1 yr.	Recurrent abscess	Large block dissection. Lateral flap graft	Results good. Recent communication
16	20	WM	1½ yrs.	Swelling first. Several abscesses	Block dissection. Packed open	Immediate results good
17	42	WM	8 yrs.	Operated 1918. Twice since. Several abscesses	Block dissection. Packed open. Midline	Results good. Recent communication
18	50	WM	2 mos.	Abscess two months before	Tract dissected out. Midline left open	?
19	22	WM	7 yrs.	Abscess, followed by several recurrent abscesses	Block dissection. Closed by method of Cantrell	Immediate results good
20	35	WM	3 yrs.	Treated for infected rectal fistula three years ago	Excision in midline. Wound packed open	?
21	25	WM	5 yrs.	Abscess. Two resections previously, followed by recurrences	Block dissection. Wound packed open	Immediate results good
22	8	WM	8 yrs.	Present at birth. Drained every two or three months. Occasional incision necessary for acute infection	Open widely. Cavity curetted and packed	Recurred in six mos. Lanced every 2 mos. since. (By letter)
23	34	WM	3 yrs.	Three years infected, draining sinus	Excision of tract. Wound packed open	Results good. Recent communication
24	30	WM	8 yrs.	Incised four times in six months. Operated by opening fistula 1932. Recurred. Incised	Wide excision of tract. Lateral flap graft	Results good. Recent communication
25	21	WM	6 mos.	Abscess formed. Opened two months later. Recurred	Extensive block dissection to coccyx. Lateral flap graft	Results good 5 months postoperative
26	16	WM	6 mos.	Abscess opened. Recurred	Block dissection with lateral flap graft	Results good five years

*Operated by several operators.

conditions warrant, should effect a cure in practically all cases. Weeder[3] feels that the more frequent removal of the coccyx is probably indicated. He placed the incidence of recurrence in the past at 25 to 35 per cent in the hands of competent surgeons. One must be radical in doing this dissection and have no thought of closure in mind during the dissection, or the operation may be inadequate.

Lahey's[4] contributions to the technic in 1929 and in 1932 were distinct advances. He removed the fear from the mind of the surgeon of leaving too large a defect. Cattell,[5] in 1934, suggested a complete closure of large defects by means of a sliding graft, according to Leahey's technic. The lateral defect was then closed in the form of a T. This gave a primary closure of both the lateral and medial defect. We mention this method and feel that although it may be successful in an occasional case, it is apt to result in disaster. Since in dealing with a large cyst, one is practically always dealing with an infected area, complete closure is very rarely feasible. In early simple cases that have very little infection, the treatment may be block dissection and simple closure, although it may be safer to leave a small drain for a short time.

It is a mistake to attempt to dissect out the fistulous tract as in the treatment of some other fistulae. It has been our observation that this has been the cause of a high per cent of recurrences. A block dissection is advisable. Frequently the coccyx should be sacrificed.

It has been the method of choice in this vicinity to inject the sinus with methylene blue. We have adhered to this method. Owen, who reported 40 cases in 1934, however, injected with iodized oil followed by x-ray noted only two recurrences. We follow our injection by block dissection and in extensive cases we have used the sliding graft with counter drainage.

One should consider injection with an opaque medium for x-ray examination. It should be kept in mind that at times the coccyx up to the sacrococcygeal junction is involved. It seems that only by careful study of the area involved and planning our surgery accordingly shall we decrease the number of recurrences.

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5. Cattell, Richard B.: Surg. Clin. N. America, **14**:1289, 1934.

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EDITORIAL DEPARTMENT

SOUTHERN MEDICAL ASSOCIATION
Thirty-Fourth Annual Meeting
Louisville, Kentucky, November 12-15, 1940

THE MEMPHIS MEETING

On the banks of the Mississippi, the second longest river in the world, in historic Memphis, has just ended the thirty-third annual convention of the Southern Medical Association. The romantic story of this city during the heyday of river traffic and travel, when many steamboats plied its channel, has been immortalized by Mark Twain. Its importance in the War-between-the-States was reflected in the bitter contest by both sides to gain and hold control of it. Memphis, now the most important commercial and medical center in a large territory embracing its own and surrounding states, gave a royal welcome to the more than four thousand medically minded guests who taxed its resources during the third week of November. Complete registration figures for Memphis show a total of 2,212 physicians with a grand total of 4,146.

On the opening day, the distinguished profession of Memphis staged a series of clinical discussions in eight groups, which were appreciated by the many early comers who attended them. These presentations represented a good cross section of Memphis medical talent. The stimulation to the city of its important medical school, the University of Tennessee College of Medicine, is evidenced by the progressiveness and scholarship of the local profession.

It is well known that no great convention such as this is ever staged by chance, but through careful planning over many months by earnest and diligent workers. The Memphis and Shelby County Medical Society, through its committees headed by Dr. W. L. Williamson, General Chairman, functioned like the timing of a modern electric clock and set an enviable example for future gatherings of the Association. The committees of women under the chairmanship of the charming and gracious Mrs. Willis C. Campbell were no less efficient in their dispensation of hospitality. The program of social and recreational entertainments was varied and entertaining to a rare degree. Never have Association members enjoyed sincerer or more delightful hospitality. The weather, like the hosts, also was kind. It remained clear and brilliant throughout the convention dates.

For the scientific programs, large general meetings were held, and meetings of specialists in large and small, inclusive and select groupings, assembled throughout the four days of activity. Their accounts of progress in medical and surgical methods in 1939 will appear in succeeding issues of the JOURNAL.

Medical motion pictures are yearly increasing in number and quality. Excellent motion picture films in Memphis demonstrated new operative and diagnostic procedures in gynecology, ophthalmology, orthopedics, medicine and other specialties, as well as the value of this method of exposition.

SCIENTIFIC EXHIBITS

Scientific exhibits were the most extensive which the Southern Medical Association has yet displayed. There were eighty-six of these demonstrations, in the study of which it would have been possible for visiting physicians to lose themselves for the duration of the convention. All outstanding exhibits cannot be mentioned. The list of those which received special awards follows. Many other fine pieces of work were seen.

First award was assigned to Dr. Graham Asher, of the University of Kansas School of Medicine, for his exhibit on lag-screen electrocardiography; second to Dr. R. H. Rigdon, of the University of Tennessee College of Medicine,

for studies on capillary permeability and inflammation; third to Dr. C. E. Sanders, of Kansas City, for his demonstration of vasoscillator and intermittent venous occlusion.

Honorable mention was given to Dr. J. L. Scianni, of the University of Tennessee College of Medicine, for his exhibit of medical art; to Drs. J. A. McIntosh, E. W. Cocke, L. F. Pierotti, A. J. Grobmyer, Jr., and M. Philonilla, of Memphis, for their exhibit on artificial fever therapy; to Drs. W. W. Waddell, Jr., DuPont Guerry, III, and McLemore Birdsong, of the University of Virginia Department of Medicine, for work on the role of vitamin K in hypoprothrombinemia and hemorrhage of the newly-born; to Drs. G. O. Broun and K. R. Andrews, St. Louis University School of Medicine, for studies on prevention of cholesterol atherosclerosis in rabbits; to Dr. Henry H. Turner, University of Oklahoma School of Medicine, for his exhibit of photographs of patients with endocrine diseases; to Drs. Willard Bartlett, Jr., St. Louis University School of Medicine, and Robert Bartlett, Washington University School of Medicine, for studies of goiter; and to Dr. Edward Chamberlain, of Temple University, Philadelphia, for his demonstration of roentgenologic studies of low back pain.

The number and diversity of exhibits in Memph's is in interesting contrast with those of the early days of the Association, and exemplifies the tremendous development of the exhibit as a means of postgraduate teaching. At the Southern Medical convention in Richmond a quarter of a century ago only two scientific exhibits were displayed, one, it is of interest to note, by an ex-president who is still a conspicuous and decorative attendant at Southern Medical meetings.

NEW OFFICERS

The Memphis meeting was presided over by the President, Dr. Walter E. Vest, of Huntington, West Virginia, who for his inaugural address gave a masterly discussion of topics now burning: the definition of a profession in contrast with a trade; methods of public health administration; the distribution of medical care for all; and an able analysis of the present tendencies toward socialization of medicine.

New officers for 1940 are: President, Dr. Arthur T. McCormack, State Health Officer, Louisville; President-Elect, Dr. Quitman U. Newell, Clinical Professor of Gynecology and Obstetrics, Washington University School of Medicine, St. Louis; First Vice-President, Dr. W. L. Williamson, Professor of Gynecology, University of Tennessee College of Medicine, Memphis; and Second Vice-President, Dr. E. W. Bertner, of Houston, Immediate Past Chairman of the Association's Section on Gynecology.

The next annual meeting will be held in Louisville, Kentucky, November 12-15, 1940.

ANTIPELLAGRIC COMPOUNDS

Thiamin and riboflavin (vitamins B_1 and B_2) are chemicals necessary for life, and an external supply of them promotes the vital processes. Accumulating evidence indicates that too narrow a significance has been attached to the role of these substances in physiologic processes, and too limited a view is taken of the presence of the deficiencies in human diets. Apparently all tissues need riboflavin and thiamin for the performance of their normal functions. Likewise, nicotinic acid is widely distributed in nature, yet it is not present in adequate amounts in the diets of many persons. Whereas no substitutes have been found for thiamin and riboflavin, a number of compounds similar to nicotinic acid in chemical formula are able to substitute for it in relieving the symptoms of human pellagra.[1] These have been discovered by various measures.

Certain strains of *B. dysenteriae* Flexner develop poorly or not at all on synthetic culture media made of a solution of several crystalline amino acids and a potassium phosphate buffer salt; but their growth is prompt on the same very simple media with the addition of nicotinic acid or amide. This fact, that nicotinic acid is the only one of the accessory food factors required by these micro-organisms, provides a new technic for study of the pharmacology and biochemical effects of the vitamin. The mode of its action in relieving pellagra is little known. If it may be investigated by observation of bacteria, which develop in twenty-four hours, instead of by using dogs, human beings, or even small laboratory animals, the subject should be more rapidly elucidated. The nature of drugs with any of the effects of the antipellagric vitamin is of interest.

The activity of twenty-four chemicals of the pyridine group, in which nicotinic acid is in-

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1. Bills, C. E.; McDonald, F. G.; and Spies, Tom D.: Antipellagric Action of Pyrazine-2, 3-Dicarboxylic Acid and Pyrazine Monocarboxylic Acid. Sou. Med. Jour., 32:793 (Aug.) 1939

cluded, in supporting growth of dysentery bacilli has recently been studied by workers at the University of Chicago.[2] Nicotinic acid, a pyridine structurally, has a benzene ring with a nitrogen atom substituting for one of the six carbon atoms and an acid group attached in a particular relationship to the nitrogen. Another product, nicotinamide, is more effective in treatment of human pellagra, canine black tongue, and in stimulating growth of dysentery bacilli. What looks to the non-chemical eye like a very slight structural change, as by moving the acid group farther from or closer to the nitrogen atom (picolinic acid and isonicotinic acid) gives a product which does not support growth of bacteria. About nine different pyridine compounds, however, support growth of *B. dysenteriae* on a synthetic medium, and roughly, so far as they have been tested, the same products are capable of curing canine black tongue.[3] A few of them have been administered to pellagrins.

Picolinic acid and isonicotinic acid, which, according to the Chicago workers,[2] do not stimulate growth of bacteria, also do not relieve canine black tongue;[3] and one of them, picolinic acid, is toxic for human beings in comparatively small doses.[1] Bills, McDonald and Spies[1] report favorable results in pellagrins following administration of two pyrazine compounds (benzene ring with two nitrogen atoms replacing two opposite carbon atoms). The two pyrazines used contained acid groups in the same relationship to nitrogen as in nicotinic acid. According to the investigators,[1] these substances are practically unknown in physiologic reactions. Their effects on bacteria or black tongue were not reported in the papers under discussion.

For dysentery bacilli, concentrations of nicotinamide up to one milligram per cubic centimeter of culture medium were beneficial. With 3, 5 and 10 milligram concentrations, there was progressive inhibition of growth. Whether these quantities in any way parallel the toxic dose for human beings might be a matter worthy of study.

It should be possible to obtain leads to analysis of vitamin action in human beings more quickly by observation of bacteria than by cure of such a disease as canine black tongue. In other words, the bacterium is a more convenient guinea pig than the dog. If the war continues, vitamin substitutes may be in greater and greater demand; and the speedier bacteriologic methods may be very useful in demonstrating new sources. In any case, the parallelism or dissimilarity of reactions of unicellular and multicellular organisms is of physiologic importance.

The stimulation of growth of certain dysentery bacilli on a synthetic medium, the cure of canine black tongue, and the relief of human pellagra are accomplished in the main, so far as investigations have gone, by the same chemical compounds.

2. Dorfman, Albert; Koser, S. A.; Reamer, H. R.; Swingle, K. F.; and Saunders, F.: Nicotinamide and Related Compounds as Essential Growth Substances for Dysentery Bacilli. Jour. Infect. Dis., **65**:163 (Sept.-Oct.) 1939.

3. Woolley, D. W.; Strong, F. M.; Madden, R. J.; and Elvehem, C. A.: Anti-blacktongue Activity of Various Pyridine Derivatives. Jour. Biol. Chem., **124**:715, 1938.

EFFECT OF A HORMONE ON THE GLAND WHICH SECRETES IT

Many drugs are now available for specific treatment of endocrine deficiency diseases: for cretinism, thyroid or thyroxin; for diabetes, insulin; for the menopause, estrogens and corpus luteum; for cryptorchidism, anterior pituitary extracts, and so on. The hormone is used clinically of course to supply a deficiency, without hope of restorative effect on the tissue from which it is derived, and treatment of hypofunction is usually continuous throughout life. The cretin must always have thyroid, the diabetic insulin.

In the case of the ovary, which functions for only a portion of a lifetime, a stimulant, anterior pituitary gonadotropic hormone, may overcome hypofunction and actually increase the internal secretion of the gland for a long period after treatment has ceased. In this sense the internal secretion may be curative, since ovarian function once initiated may continue in the normal rhythm without continuation of anterior pituitary injections. Functioning of the ovary, with cellular differentiation of its tissue, may thus be induced by a specific medicament.

Endocrine hypofunction may be treated by supplying the deficient secretion; treatment of hyperfunction in clinical practice is usually specifically by surgery, or removal of all or a part of the offending gland. Medication of hyperfunction is in a much more confused state.

The anterior pituitary gland is considered the regulator or motor of many endocrines; reverse effects of these other glands upon the pituitary are less accepted. Estrogens from the ovary are

believed in some quarters to stimulate the anterior pituitary; in others to inhibit it. Lauson,[1] of the University of Wisconsin, studied the anterior pituitaries of young rats which had received injections of anterior pituitary gonadotropic hormone. He induced sexual precocity in immature animals by this treatment, and after the ovaries had enlarged to a certain weight, estimated the hormone content of the anterior pituitaries. As the precocious ovaries increased in size, the gonadotropic hormone of the pituitary was regularly reduced in quantity. The relationship of anterior pituitary gonadotropic hormone content and ovary size was inversely proportional; as ovarian secretion increased, gonadotropic hormone decreased to a certain point, that is, to less than half the normal content.

The reverse effect has also often been noted: that the anterior pituitaries of castrate animals contain more gonadotropic hormone than the normal. Gonadotropic hormone goes down as ovarian hormone goes up, and up as ovarian hormones go down.

The reduction of anterior pituitary gonadotropic content after injections of the hormone may be interpreted as mediated through the ovaries. Whether this is or is not the mechanism must be a matter of conjecture since many other factors are involved.

Since Seale Harris'[2] report on hypo-insulinism, it has been generally accepted that any endocrine disease may manifest itself clinically in two ways. It may result from either hyper- or hypo-activity of the organ involved. With the purification of numerous internal secretions which has been accomplished in the past twenty years, treatment of under function has been increasingly successful.

It has been noted that ovarian activity and formation of follicles are inhibited by treatment with estrogenic (ovarian) material. The ovary may assume a smooth inactive appearance in a patient or animal which has received ovarian hormone. This inactivating effect may be interpreted as coming through the pituitary, and as meaning that ovarian hormone inhibited the anterior pituitary which in turn failed to stimulate the ovary. However attained, the result physiologically is reduction of ovarian function by treatment with ovarian hormone; and from the quoted work of Lauson, reduction of anterior pituitary activity by treatment with anterior pituitary hormone. It would seem to be a sort of hair-of-the-dog-is-good-for-the-bite effect.

From the physiologist's point of view, it should be possible to depress overactivity of the thyroid with thyroid hormone, and to reduce pancreatic islet tumors with insulin. Therapy of this type will not be attempted because of the probable immediately dangerous effects of increasing the circulating hormone in such conditions.

Physiologically the hypothesis is quite tenable that the secretion of an endocrine gland specifically inhibits the secretory activity of that tissue. Glandular depression may be brought about by the secretion of the particular gland. Gland stimulation must come from another source.

1. Lauson, Henry D.: The Gonadotropic Content of the Female Rat Hypophysis in Experimental Precocious Puberty. Amer. Jour. Physiol., **127**:629 (Nov.) 1939.
2. Harris, Seale: J.A.M.A., **83**:729, 1924.

TWENTY-FIVE YEARS AGO

From Journals of 1914

The War.—The king spent nearly the whole of last week with his troops in France. * * * Sir Berkeley Moynihan left for France on December 4 to take up his appointment as Consulting Surgeon with the British Force Overseas.[1]

Army's Teeth.—British Red Cross Hospital, Rouen, December 12. The arrival of the first dental officers for service with the troops in France was welcome, for they were very much needed. * * * There is a wise provision of the authorities that sergeants and some others shall have a right to artificial teeth.[2]

Care of the Wounded.—The healing power of the winds of the Heavens is now established beyond all question; but is there not also virtue during winter's stormy nights in the peace and cheeriness of a hot dinner, an arm chair, and a blazing fire? A return to the latter after the "open air hospital" at Cambridge (which has undergone no modification since its inauguration in the dog days) brings wonderings as to what proportion of our medical profession would really approve of the "open air treatment" at one of the dampest spots in England of those 1,250 brave defenders of our hearths and homes who have been sent back from the trenches, wounded and suffering, into the heart of old England at Christmas time, only to land in a cricket field, now one vast muddy slosh, across which are stretched rows of draughty open verandahs that constitute the hospital! No heating arrangements whatever, no tables, no chairs. * * * Nurses in goloshes and with hands all chilblains wrestling with the elements for possession of surgical dressings. * * * The cases do well and the total results seem as good as those from any other hospital. * * * Why, if the healing results are not markedly superior, should lads with terri-

1. The War. Lancet, **187**:1387 (Dec. 12) 1914.
2. Newland-Peley, F.: Our Army's Teeth. Ibid., p. 1430 (Dec. 19) 1914.

ble injuries be deprived of any harmless and obtainable comfort?[3]

Tetanus.—The treatment of tetanus properly engages the attention of the medical world at present. * * * Dr. Caillaud and Dr. Corniglion * * * had recourse to * * * injections of phenol to which were added lantol or colloidal rhodium. The phenol was in a 1 per cent solution and intramuscular injection of 40 c. c. to begin with was given daily * * * increasing concentration. The four cases thus treated were cured.[4]

Infected Wounds.—This is an artillery war in which shrapnel does the damage, tears flesh, breaking bones, and always causing jagged irregular wounds * * * the great tragedy, sepsis, everywhere, unavoidable sepsis. * * * At first there was not sufficient [tetanus] antitoxin, but now it is given at the front as early as possible * * * the other infection is more serious, an emphysematous wound gangrene, due to an anaerobic gas bacillus.[5]

3. Sayer, Ettie: Open Air Treatment for Wounded Soldiers. Lancet, **187**:1495 (Dec. 26) 1914.
4. Paris Correspondent. Treatment of Tetanus. Lancet, **187**: 1382 (Dec. 12) 1914.
5. Osler, Sir William: Correspondence. J.A.M.A., **63**:2303 (Dec. 26) 1914.

Correspondence

TOXICITY OF STILBESTROL

Editor, SOUTHERN MEDICAL JOURNAL:

I read with interest your editorial on toxicity of drugs in the September issue of the JOURNAL. For the last nine months I have been running complete blood counts, sedimentation tests, blood chlorides and nonprotein nitrogen determinations on patients under treatment with diethyl stilbestrol, some of whom have received as much as 1,000 milligrams. There has been no change in the values of the above tests. With 16 gynecologic patients in the Jefferson Davis Hospital under complete control, a complete blood count, sedimentation and nonprotein nitrogen estimation were made every other day. The patients were given 25 milligrams every other night until 200 to 1,000 milligrams were taken, with no change in the laboratory tests.

From this study I think stilbestrol is not toxic in the doses recommended for the treatment of menstrual disorders.

The only harmful effects observed have been in cases of amenorrhea and the menopause, in which the patients took too many tablets over too long a period. All of these began to bleed so profusely that they had to enter the hospital and have a dilatation and curettage. This drug should be given in small amounts, the patient to be instructed to discontinue it if bleeding begins. This refers to cases of amenorrhea, hypomenorrhea and menopause, where small doses of stilbestrol are given, 0.1 to 1 milligram daily.

As to nausea and vomiting: bile acids and salts and the taking of the stilbestrol tablets at bedtime or better at about six in the afternoon, will usually eliminate nausea. Ninety-five per cent of patients have become tolerant to stilbestrol by the fifth or seventh day.

We have ovaries or sections of ovaries from 24 patients who received 25 to 1,000 milligrams of stilbestrol over a period of 12 hours to 30 days. In all, the follicle cysts were obliterated and the ovary became smooth like the ovary of a pregnant woman at term. Stilbestrol therefore caused a physiologic curettement and eliminated ovarian follicles better than can be done with a knife by resection or by puncturing cysts. After stilbestrol is discontinued, the ovary begins anew and in many cases the menstrual cycle returns to normal.

We are conducting further studies on the toxicity of stilbestrol.

KARL JOHN KARNAKY, M.D.
Houston, Texas, October 26, 1939.

Book Reviews

Primer of Allergy: A Guidebook for Those Who Must Find Their Way Through the Mazes of This Strange and Tantalizing State. By Warren T. Vaughan, M.D., Richmond, Virginia. 140 pages, illustrated. St. Louis: The C. V. Mosby Company, 1939. Cloth $1.50.

Dr. Vaughan's former book on allergy was written in the form of a questionnaire with answers attached. It was for use by physicians and patients alike. Recently he has divided his material into two books. The first is a large one entirely for the physician. The second is this small "Primer of Allergy" which he has written for lay distribution.

"Primer of Allergy" is written in everyday English with few long or scientific terms. It is illustrated with numerous cartoons which have appeared in the lay press. Dr. Vaughan makes frequent use of simile and metaphor to clarify his meaning. This little book is written for the purpose of giving the lay sufferer from allergy a clear understanding of his affection. For this purpose it is excellently adopted. It should likewise be easy reading for any physician who wishes a simple understanding of this interesting and complicated subject.

Diseases of the Foot. By Emil D. W. Hauser, M.S., M.D., Assistant Professor of Bone and Joint Surgery, Northwestern University Medical School, with a Foreword by Sumner L. Koch, M.D. 472 pages, illustrated. Philadelphia: W. B. Saunders Company, 1939. Cloth $6.00.

This is a comprehensive book, which will serve as a basis for presenting the anatomy and physiology of the normal and abnormal foot during the period of growth and development. It also serves as a guide to the treatment of foot disorders as they appear in infancy, and as they develop during the period of rapid growth and in adult life. It is well written, thorough and profusely illustrated. Fractures, dislocations, circulatory disturbances and tumors are covered, in addition to static disturbances. All established operative procedures are adequately described.

This presentation of diseases of the foot is the most complete so far published in the English language.

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Southern Medical News

ALABAMA

The Jefferson County Medical Society has opened its new auditorium in the new Hillman Clinic Building, Birmingham. The auditorium has a seating capacity for three hundred and an adjoining club room. There are provisions in the auditorium for a loud speaker, and included in the furnishings are a talking moving picture machine and a stationary screen.

Dr. Walter J. Broad, Montgomery, has recently been appointed Health Officer of Bullock County to succeed Dr. Hubert R. Owen, Union Springs, who resigned to do postgraduate work at the University of Pennsylvania.

Dr. Benjamin S. Black, Grove Hill, has resigned as Health Officer of Clarke County.

Dr. James Edwards Cameron, Alexander City, and Miss Edith Ellison, Hurtsboro, were married August 1.

Dr. William J. Donald, Brewton, and Miss Vivian Frazier, Mobile, were married August 4.

Dr. Josiah Harris Smith, Selma, and Mrs. Gladys Buckmaster Wire, Cincinnati, Ohio, were married in August.

Dr. William Ernest Coleman, Jr., Birmingham, and Miss Betty Brown, Eastaboga, were married September 30.

Deaths

Dr. William Yancey White, Center, aged 78, died August 11 of coronary occlusion.

Dr. Floyd B. Moore, Fairhope, aged 71, died August 5 of chronic myocarditis and arteriosclerosis.

Dr. Edwin C. Bandy, Alabama City, aged 78, died in September of extensive burns and bronchopneumonia.

Dr. Winston W. Barnard, Headland, aged 79, died August 22 of a carbuncle on the neck and chronic myocarditis.

Dr. Arthur Wellington Ligon, Oxford, aged 80, died August 18.

ARKANSAS

The Tenth Councilor District Medical Society has elected the following officers for the coming year: President, Dr. B. L. Ware, Greenwood; Vice-President, Dr. J. W. Amis, Fort Smith; Secretary-Treasurer, Dr. Ruth Ellis Lesh, Fayetteville.

The Second Councilor District Medical Society has elected the following officers for the coming year: President, Dr. A. H. Hudgins, Searcy; Vice-President, Dr. M. S. Craig, Batesville; Secretary-Treasurer, Dr. O. J. T. Johnston, Batesville.

The First Councilor District Medical Society has elected the following officers for the coming year: President, Dr. F. H. Jones, Piggott; Vice-President, Dr. E. B. McKelvey, Paragould; Secretary, Dr. J. H. McCurry, Cash, re-elected.

Dr. Raymond L. Gregory, formerly Professor of Medicine at Howard University College of Medicine, Washington, D. C., has been appointed Professor and Head of the Department of Medicine at the University of Arkansas School of Medicine, Little Rock.

Dr. Fred H. Krock, Fort Smith, did special work at the Mayo Clinic during the month of October.

Dr. H. K. Carrington and Dr. J. H. Wilson have opened their new clinic at Magnolia.

Dr. J. B. Askew, Batesville, and Dr. Kirk Mosley, Texarkana, are taking public health work at Harvard University.

Dr. R. B. Robins, Camden, took special work in proctology recently in Chicago.

Dr. O. K. Hukill has moved from Hot Springs National Park to Walnut Ridge.

Dr. A. A. Hughes has moved from Pine Bluff to Houston, Texas.

Dr. J. L. Aday, Little Rock, did postgraduate work at the Mayo Clinic in September.

Continued on page 35

The Tulane University of Louisiana
SCHOOL OF MEDICINE

POSTGRADUATE instruction in all branches of medicine is offered to graduate physicians.

Review Courses in medicine, in surgery and in gynecology and obstetrics begin January 8, 1940, and continue through March 30, 1940. Three courses of four weeks each are offered in each branch.

A special course in surgical technic will begin January 8, 1940.

A special course in gynecological surgery will be offered beginning January 8, 1940.

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Washington University School of Medicine

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OTOLARYNGOLOGY

An eight months' course in Otolaryngology for those beginning the study of diseases of the ear, nose and throat.

Only graduates of medical schools who have completed a year of intern service will be admitted to the course.

The course begins September 23, 1940. Fee for the course is $600.00.

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The Registrar
WASHINGTON UNIVERSITY SCHOOL OF MEDICINE
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(*See back of this page*)

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—SOUTH. M. J., *23:*1092, December, 1930

"*Roentgenographic studies of the paranasal sinuses should be made in all cases of arthritis, regardless of whether or not the patient has nasal symptoms.*"
—M. CLIN. NORTH AMERICA, *21:*1683, November, 1937

"*During the past several years it has been my custom to make at least one or two roentgenograms of the accessory sinuses of patients of all ages sent to me for study of the lungs in whom I could find no evidence of tuberculosis, but who showed evidence of other infectious processes. Sinus disease has been found in a surprisingly large number.*"
—ANN. INT. MED., *9:*550, November, 1935

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(*See reverse side of this page*)

Continued from page 34

Dr. Edward Adams, Hazen, has moved to DeValls Bluff.

Dr. C. B. Dixon, Kingston, has moved to Decatur.

Dr. T. J. Cunningham, Jr., Pine Bluff, and Miss Margaret Davis, Rosston, Louisiana, were married September 18.

Deaths

Dr. James Homer Buckley, Fort Smith, aged 64, died July 31 of heart disease.

Dr. Joseph Lafayette Minton, Hartman, aged 85, died August 7 of pernicious anemia.

Dr. J. O. Bickham, Winchester, aged 58, died August 13 of myocarditis, acute dilatation of the heart, nephritis and hypertension.

Dr. Edwin Justus Haster, Dardanelle, aged 30, died September 24.

Dr. Ervin Layman Matthews, Morrilton, aged 54, died October 17.

Dr. Thomas C. Neese, Walnut Ridge, aged 72, died October 17.

DISTRICT OF COLUMBIA

Dr. Malcolm D. Harrison, Washington, has been appointed Director of the Outpatient Department of Garfield Memorial Hospital.

Dr. Robert Beall Hightower, Washington, and Miss Rossell MacDonald, North, Virginia, were married July 20.

Death

Dr. John William Mann, aged 45, died September 28 from a heart attack.

FLORIDA

The Florida East Coast Medical Association has elected the following officers for the coming year: President, Dr. I. M. Hay, Melbourne; First Vice-President, Dr. E. C. Swift, Jacksonville; Second Vice-President, Dr. A. J. Logie, Jacksonville; Secretary-Treasurer, Dr. J. S. Stewart, Miami. The 1940 meeting of the Association will be held in Miami.

Dr. Thomas E. Buckman, Jacksonville, has been appointed Medical Director of the King Edward Industrial Nursery, which is said to be the nation's first industrial nursery.

Dr. Hollis C. Ingram, Orlando, took a special course in nose and throat work at Cook County Graduate School of Medicine in Chicago during September.

Dr. Ralph E. Russell, Ocala, is doing special work in ophthalmology at the Cook County Graduate School of Medicine in Chicago.

Dr. R. B. Harkness, Lake City, recently received Fellowship in the American College of Surgeons.

Dr. Waldo Horton, Winter Haven, did special work in dermatology and syphilology at Charity Hospital in New Orleans during the month of August.

Dr. Donald W. Smith and Dr. Perry D. Melvin, both of Miami, spent the month of September in Philadelphia doing postgraduate work.

Dr. J. E. Harris, Sarasota, did postgraduate work in cardiology during the month of September at the Massachusetts General Hospital, Boston.

Dr. M. B. Marks, Miami Beach, has returned after taking a course in allergy at Harvard University and Massachusetts General Hospital.

Dr. W. J. Williams, DeLand, has moved to Cassia.

Dr. J. H. Chiles, Holopaw, has moved to Orlando.

Dr. James A. Bradley and Mrs. Evelyn Neuling, both of St. Petersburg, were married October 1.

Continued on page 36

THE NEW YORK POLYCLINIC
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UROLOGY

A combined full time course in Urology, covering an academic year (8 months). It comprises instruction in pharmacology; physiology, embryology; biochemistry; bacteriology and pathology; practical work in surgical anatomy and urological operative procedures on the cadaver; regional and general anesthesia (cadaver); office gynecology; proctological diagnosis; the use of the ophthalmoscope; physical diagnosis; roentgenological interpretation; electrocardiographic interpretation; dermatology and syphilology; neurology; physical therapy; continuous instruction in cysto-endoscopic diagnosis and operative instrumental manipulation; operative surgical clinics; demonstrations in the operative instrumental management of bladder tumors and other vesical lesions as well as endoscopic prostatic resection.

FOR INFORMATION ADDRESS

MEDICAL EXECUTIVE OFFICER, 345 West 50th Street, NEW YORK CITY

Continued from page 35

Dr. James Nelson Patterson and Miss Viola Townsend Davis, both of Jacksonville, were married September 30.

DEATHS

Dr. Paul Eskeberg, Miami, aged 28, died September 3 of chronic myelogenous leukemia.

Dr. Benjamin Franklin Hawk, West Palm Beach, aged 72, died August 15 of carcinoma of the prostate.

Dr. Berton Mell Bishop, Archer, aged 70, died August 22 of organic heart disease.

Dr. Joseph Lee Kirby-Smith, Jacksonville, aged 57, died November 5 following a brief illness.

GEORGIA

Atlanta will be host to the annual meeting of the Radiological Society of North America, December 11 to 15.

Dr. James H. Litton, formerly of Carbon, West Virginia, has succeeded Dr. Stuart P. Vandiviere, Milledgeville, as Commissioner of Health for Baldwin County.

Dr. George M. Anderson, Morgan, has succeeded Dr. Luther A. Brendle, Eastman, as Commissioner of Health for Dodge County.

Dr. Oliver W. Jenkins, Edison, has succeeded Dr. Clair A. Henderson, Dawson, as Commissioner of Health for Sumter County.

Dr. Robert H. Haralson, Jr., Tifton, has resigned as Health Commissioner of Tift County to enter private practice.

Dr. M. E. Groover, Quitman, Commissioner of Health for Brooks County, is taking an eight months' postgraduate course in health work at Johns Hopkins University School of Medicine, Baltimore.

The Georgia Urological Association has elected the following officers for the coming year: President, Dr. M. K. Bailey, Atlanta; President-Elect, Dr. J. L. Garrard, Rome; Secretary-Treasurer, Dr. W. E. Upchurch, Atlanta.

Dr. Needham Bryant Bateman and Miss Florence Estele Stevenson, both of Atlanta, were married September 14.

Dr. Lewis E. Abram, Fitzgerald, and Miss Doris Friedman, Madison, were married September 10.

DEATHS

Dr. Arthur Godfrey Fort, Atlanta, aged 61, died September 15 of myocarditis and pulmonary edema.

Dr. James Wilmarth Clark, Columbus, aged 56, died August 24 of cerebral hemorrhage.

Dr. Joseph H. Bradfield, Atlanta, aged 72, died September 6.

Dr. William Bryan Summerall, Atlanta, aged 74, died September 5 of heart disease.

Dr. Roy S. Pounds, Redan, aged 50, died September 18.

Dr. Parish Stewart Smith, Conyers, aged 62, died September 7.

Dr. Newton J. Boswell, Decatur, aged 66, died September 8 of cerebral hemorrhage, arteriosclerosis and hypertension.

Dr. James J. Kennedy, Collins, aged 78, died September 11.

Dr. W. K. Burnett, Winston, aged 70, died September 20 from heart disease.

Dr. Thomas W. Taylor, LaGrange, aged 75, died November 1 following a brief illness.

KENTUCKY

Dr. Elmer R. Schnake, Newport, has been appointed Health Officer of that city.

Dr. George B. Davis, formerly of Guilford, Connecticut, succeeds Dr. Chester R. Markwood, of Glasgow, as Health Officer of Barren County.

Dr. Reuben M. Coblin, Frankfort, has been appointed Health

Continued on page 38

Intensive Post-Graduate Course in Ophthalmology

The George Washington University School of Medicine, Washington, D. C.

For graduates in medicine, March 25-30, 1940, inclusive

Drs. A. Bielschowsky, Wiley R. Buffington, Grady E. Clay, Harry S. Gradle, Daniel B. Kirby, Peter Kronfeld, Ralph I. Lloyd, S. Hanford McKee, C. S. O'Brien, Avery DeH. Prangen, Bernard Samuels, Albert C. Snell, Edmund B. Spaeth, Georgiana Dvorak Theobald, Philips Thygeson, Robert Von der Heydt, and Mr. Scott Sterling.

Resident Members: Drs. Wm. Thornwall Davis, Ernest Sheppard, G. Victor Simpson, E. Leonard Goodman, Ronald A. Cox, Frank D. Costenbader, Horace E. Allen, Commander J. E. Adams, M.C. USN; Lt. Col. J. E. Ash, M.C. USA; Lt. Col. Frederic H. Thorne, M.C. USA; Lt. Col. Virgil H. Cornell, M.C. USA.

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SPECIAL PRACTICAL 5-DAY COURSE limited to 25 participants, March 19-23rd, 1940, inclusive, on OCULAR SURGERY, PATHOLOGY AND ORTHOPTICS

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Interviews regarding Board Examinations will be given by members of the American Board of Ophthalmology to those registered for the course.

For further details apply to the secretary, Miss Louisa G. Wells, 927 17th St., N.W., Washington, D. C.

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Continued from page 36

Officer of Franklin County to succeed the late Dr. Eugene C. Roemele.

Dr. James W. Miller, Greensburg, has resigned as Director of Green County Medical Society to enter private practice.

Dr. William Ray Moore and Miss Elizabeth Anderson Mason, both of Louisville, were married September 8.

Dr. Frank M. Warder, Glasgow, and Miss Irene Katherine Randolph, Detroit Lakes, Minnesota, were married August 11.

DEATHS

Dr. William O. Bailey, Louisville, aged 76, died August 4 of cerebral hemorrhage.

Dr. Harry Thompson Liggett, Louisville, aged 64, died August 17 of staphylococcic septicemia.

Dr. Abraham B. Deany, Winchester, aged 62, died August 13 of myocarditis.

Dr. Robert F. Peak, Louisville, aged 80, died August 16 of hypertrophy of the prostate and heart disease.

LOUISIANA

Dr. Rupert E. Arnell, Clinical Professor of Obstetrics and Gynecology, and Dr. William F. Guerriero, Instructor in Obstetrics and Gynecology, and Dr. Philips J. Carter, Professor of Obstetrics and Gynecology and Co-Director of the Department in the School of Medicine of Louisiana State University, have recently been engaged in giving postgraduate instruction in obstetrics in the northern part of Louisiana.

Dr. Erasmus D. Fenner, New Orleans, recently presented the Library of the Louisiana State University School of Medicine a collection of medical books and journals from his library.

Dr. Edgar Hull, New Orleans, formerly Assistant Professor of Medicine, Louisiana State University School of Medicine, has been appointed acting Head of the Department of Medicine to succeed the late Dr. George S. Bel.

Dr. Carlo J. Tripoli, New Orleans, has been appointed Professor of Medicine in charge of the Department of Medicine of the Graduate School of Louisiana State University.

Dr. John R. Schenken, New Orleans, has been appointed Assistant Dean of the School of Medicine of Louisiana State University.

Dr. Roy B. Harrison, New Orleans, has been named a member of the Advisory Council on Medical Education, succeeding the late Dr. Harold Ryrins.

Dr. Charles H. Mosely, Jr., Monroe, and Miss Florence Jane Morley, Rayville, were married August 5.

DEATHS

Dr. Maurice Joseph Gelpi, New Orleans, aged 56, died August 9 of cardiovascular disease and cerebral hemorrhage.

Dr. Harper Leonidas Crow, Bossier, aged 53, was killed September 3, when the truck in which he was driving overturned.

Dr. Julius E. Isaacson, New Orleans, aged 45, died October 17.

MARYLAND

Dr. Daniel H. Kress and his wife, Dr. Lauretta E. Kress, recently entertained with a farewell gathering on the lawn of the Washington Sanitarium, Takoma Park, where they have served on the staff for thirty-two years. They will retire to Orlando, Florida.

Dr. William Curtis Stifler, Jr., and Dr. Jean Evans Rose, both of Baltimore, were married July 29.

Dr. Ferdinand E. Chatard, IV, Baltimore, and Miss Constance Bentley Lyon, Purcellville, Virginia, were married September 9.

DEATHS

Dr. Louis Mervin Maus, Rockville, aged 88, died August 3 of obstructive jaundice.

Continued on page 40

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THIS LITTLE GIRL WILL HAVE A HAPPY CHRISTMAS but

Tuberculosis is still the greatest killer of youth . . . and takes *fifty per cent more girls* than boys between the ages of 15 and 25!

Two modern aids that help the physician detect tuberculosis in its earliest, *curable* stage are the tuberculin test and the chest X-ray.

Your purchases of Christmas Seals make it possible, not only to teach people that tuberculosis is preventable and curable, but to look for early stages of this dread disease among children who *seem to be* in good health.

So from now 'til Christmas, mail no letter—send no package—unless it is decorated with the Christmas symbol that saves lives!

The National, State and Local Tuberculosis Associations in the United States

1939 **BUY CHRISTMAS SEALS**

Continued from page 38

Dr. Jefferies Buck, Baltimore, aged 68, died August 14 of arteriosclerosis and hypertensive cardiovascular disease.

Dr. Cooper Curtice, Beltsville, aged 83, died August 8 of coronary occlusion.

Dr. John H. T. Earhart, Westminster, aged 77, died September 10 of coronary thrombosis.

Dr. George Wallace Jarman, Princess Anne, aged 77, died September 19 of acute myocarditis.

MISSISSIPPI

Deaths

Dr. Frank Henry Hagaman, Jackson, aged 43, died August 19 of injuries received in an automobile accident.

Dr. Ben C. Crisler, Aberdeen, aged 62, died August 10 of poisoning.

Dr. Henry A. Smith, Sumner, aged 81, died August 1 of coronary occlusion.

Dr. Randall Schuyler, Long Beach, aged 80, died recently.

MISSOURI

Dr. Evarts A. Graham, St. Louis, was recently honored on his twentieth anniversary as Professor of Surgery at Washington University School of Medicine. A two-day scientific program was presented by men who were former students of Dr. Graham's dealing with the work they are doing at present.

Dr. H. L. Mantz, Kansas City, has recently been made a member of the governing council of the Mississippi Valley Conference on Tuberculosis.

Dr. Joseph Boisliniere Grindon, Jr., St. Louis, and Miss Mary Ann Murray, Carlyle, Illinois, were married September 27.

Deaths

Dr. John Harrison Timberman, Chillicothe, aged 63, died August 22 of cerebral hemorrhage.

Dr. William L. Turner, Springfield, aged 68, died August 30 of injuries received in a fall.

Dr. Samuel A. Benson, St. Louis, aged 72, died September 3 of burns received when his home caught fire.

Dr. Samuel L. Bates, Adrian, aged 87, died August 24 of pneumonia.

NORTH CAROLINA

The Duke University School of Medicine, Durham, recently conducted its sixth annual symposium. The title of the symposium was "Diseases of the Lungs and Thorax."

Dr. R. B. McKnight, Charlotte, was recently elected to active membership in the American Association for the Study of Goiter at the meeting of the Society in Cincinnati.

Dr. Frank Jones, at present doing graduate work at Johns Hopkins Hospital in Baltimore, has been appointed Surgeon to the Catawba General Hospital, Newton.

Dr. Meyer Vitsky, Richmond, announces the removal of his office to 1108 West Franklin Street, practice limited to obstetrics and gynecology.

Dr. Frank Allen Jones, a native of Cofield, who is serving his internship at the University of Pennsylvania Graduate Hospital, was recently awarded a certificate of Exemplary Service and a fifty-dollar cash prize by the institution.

Dr. Aubrey Hawes announces the opening of offices in the Medical Arts Building, Charlotte; for practice limited to urology and genito-urinary surgery.

Dr. Philip Naumoff announces the opening of offices at 706 Independence Building, Charlotte, for the general practice of medicine.

Dr. Fred G. Patterson, Chapel Hill, and Miss Julia Baylor Shirley, Richmond, were married September 5.

Dr. Herman W. Farber, Weldon, and Miss Henrietta Salsbury, Richmond, Virginia, were married August 30.

Dr. John D. Fitzgerald, Roxboro, and Miss Betty Kathlyn Offerman, Durham, were married September 5.

Dr. Franklin LeRoy Wilson, Montreat, and Miss Eleanor D. Garrett, Miami, Florida, were married September 7.

Dr. Warner Lee Wells, Raleigh, and Miss Rebecca Ann Atzrodt, Clarksburg, West Virginia, were married October 7.

Deaths

Dr. Joseph Hubbard Saunders, Williamston, aged 56, died August 3 of hepatitis.

Continued on page 42

Schering Presents

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PRANONE offers the physician a synthesized substance (chemically closely related to the corpus luteum hormone, progesterone) which, administered orally, exerts progesterone-like action. PRANONE is pure crystalline anhydro-hydroxy-progesterone in tablet form. The physician now has in PRANONE tablets an oral treatment of corpus luteum hormone deficiencies as effective as Proluton injections.

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COMPLETE INFORMATION AND LITERATURE AVAILABLE FROM THE MEDICAL RESEARCH DIVISION

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SCHERING CORPORATION
BLOOMFIELD, NEW JERSEY

Continued from page 40

Dr. Robert Lee Cater, Jr., Greensboro, aged 44, died August 8 of acute dilatation of the heart.

Dr. James Cannon Greene, Greenville, aged 70, died August 15 of chronic myocarditis.

Dr. Napoleon B. Houser, Charlotte, aged 69, died August 28 of cerebral hemorrhage.

Dr. Howard Leon Sumner, Asheville, aged 37, died August 16 of heart disease.

OKLAHOMA

Dr. Richard M. Adams, Ada, has been appointed Health Officer of Tulsa.

Dr. Solomon P. Roberts, Nowata, has recently been appointed Health Officer of Nowata County.

Dr. William C. Tisdale, Medical Superintendent of the Western Oklahoma Tuberculosis Sanatorium, Clinton, has resigned to resume private practice.

Dr. Joe Dorrough, Haileyville, has recently been appointed Health Superintendent of Pittsburgh County.

Dr. C. C. Gardner, Ponca City, has been appointed temporary Superintendent of Health of Kay County by the State Health Department.

Dr. Ray H. Lindsey, Pauls Valley, recently did postgraduate work at the Cook County Hospital in Chicago.

Dr. John F. Park, McAlester, has returned after visiting surgical clinics in London, England.

Deaths

Dr. Graham Wall Diggs, Wetumka, aged 59, died August 18.

Dr. Alden J. Brace, Vici, aged 66, died August 7 following an operation for ruptured appendix.

Dr. James L. Shuler, Durant, aged 79, died August 24 of chronic nephrosis.

Dr. Caroline LeMonte Rolph Bassmann, Claremore, aged 70, died September 30.

Dr. Walter S. Stevens, Oklahoma City, aged 55, died September 11 following an extended illness.

Dr. Virgil May Wallace, Morris, aged 58, died August 22 following a stroke of paralysis.

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Doctors admitted at any time for review and clinical observation.

OSCAR B. NUGENT, M.D., Director

Dr. L. A. McComb, Tulsa, aged 45, died September 2.

SOUTH CAROLINA

The following South Carolina physicians were added to the membership roster of the American College of Surgeons at the recent annual meeting in Philadelphia: Dr. Purvis J. Boatwright, Orangeburg; Dr. John G. Cathcart, Gaffney; and Dr. Everett Eldred Herlong, Rock Hill.

The Anderson County Tuberculosis Sanitarium Association recently elected the following officers: President, Dr. Herbert H. Harris, Anderson; Vice-President, Dr. D. C. Stoudemire, Honea Path; Treasurer, Dr. Grady Clinkscales, Anderson.

Dr. Leon H. Banov, Charleston, was elected President of the International Society of Medical Health Officers at the recent meeting in Pittsburgh.

The Medical Society of South Carolina (Charleston County) will celebrate on December 5 the founding of the Charleston Medical Society one hundred and fifty years ago. Many special features and eminent speakers will honor this colorful celebration.

Dr. Clarence Alexander Kinney, Florence, and Miss Rosalie West Parks, Bennettsville, were married August 26.

Dr. Robert Lee Sanders, Columbia, and Miss Helen Jessa McDowell, Charleston, were married September 23.

Deaths

Dr. Homer Bowen, Waltersboro, aged 52, died August 26 of cerebral hemorrhage.

TENNESSEE

Dr. Jewell R. Wilson has resigned as Superintendent of the Western State Hospital.

Dr. James E. Dedman, Manager of the Veterans' Administration Facility, Memphis, has taken a leave of absence, being succeeded by Dr. Horace C. Dodge, recently of Fort Bayard, New Mexico. Dr. Dedman will retire after January 31, 1940, when he will have reached the retirement age.

Dr. William S. Muse, Lexington, and Miss Loretta Greaney, Worcester, Massachusetts, were married September 2.

Dr. George Kenneth Scholl and Miss Dorothy Ophelia Rhea, both of Johnson City, were married August 25.

Dr. James Morris Higginbotham and Miss Sue Dobbins, both of Chattanooga, were married September 6.

Dr. E. V. Pollard has r.moved from Knoxville to Chicago, Illinois.

Dr. M. M. Satterfield has moved from Memphis to Trezevant.

Dr. John A. Conroy has moved from Sevierville to Cambridge, Massachusetts.

Dr. A. E. Hardison has moved from Maryville to Nashville.

Dr. Milton C. Wiggins has moved from Paris to Tucson, Arizona.

Dr. W. O. Vaughan, Nashville, announces the removal of his office to 1913-1915 Church Street.

Dr. Sam C. Cowan, Jr., Nashville, announces the opening of offices at 306 Doctors Building for the practice of obstetrics and gynecology.

Dr. Robert M. Jeter, Nashville, has moved to Gleason.

Continued on page 44

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Discriminating Physicians are Prescribing

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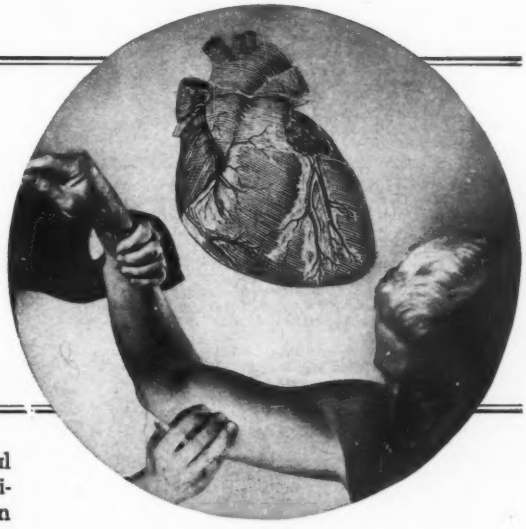
Conserving ARTERIAL ELASTICITY



● The conservation of arterial elasticity for as long as possible is an important objective in the treatment of hypertension. Radical measures are applicable only to a small number of patients. In the majority, regulation of diet is instituted and the need for avoidance of physical overexertion and emotional excitement is stressed.

Supplementary to these fundamental therapeutic measures, many physicians prescribe Theominal, a logical form of therapy because the reduction of blood pressure which follows its use is gradual and never precipitate. Furthermore, Theominal relieves and often prevents mental stress and strain and thus helps to guard the patient against vascular crises.

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LOUISVILLE, KENTUCKY

Continued from page 42

Dr. J. H. Lipsey, Memphis, has moved to Brookhaven, Mississippi.

Dr. T. W. Jones, Martin, has moved to Bradford.

DEATHS

Dr. John H. Buchanan, Memphis, aged 33, died November 9 from injuries received in an automobile accident.

Dr. Robert Logan Jones, Nashville, aged 68, died September 15.

Dr. A. C. Elinor, McKenzie, aged 64, died September 15.

Dr. Henry LeHardy, Chattanooga, aged 86, died August 15.

Dr. James G. Robertson, Arrington, aged 77, died August 21 of carcinoma of the right side of the jaw.

Dr. S. W. Williams, Gassaway, aged 58, died August 19 of coronary embolism.

Dr. Nathan Easterly Hartsook, Johnson City, aged 70, died August 10 of coronary thrombosis.

TEXAS

Dr. D. R. Venable, Wichita Falls, spent the months of August and September at Mayo Clinic, Rochester, Minnesota, studying tissue pathology.

Dr. P. E. Fish, Electra, has returned after doing postgraduate work in Rochester, Minnesota.

Dr. L. P. Hightower, Fort Worth, is doing postgraduate work in allergy at the University of California.

Dr. Charles R. Williams, Mineral Wells, recently did postgraduate work at the University of Colorado.

Dr. T. D. Young, Roscoe, is doing postgraduate work in Rochester, Minnesota, and Chicago, Illinois.

Dr. T. C. Terrell, Fort Worth, is doing postgraduate work in cardiology at Harvard University in Boston.

Dr. W. W. Beach has moved from Lubbock to Littlefield.

Dr. H. J. Bonstetter has moved from Cibola to Houston.

Dr. E. Truett Crim has moved from Dallas to Greenville.

Dr. R. L. Baily has moved from Dallas to Wichita Falls.

Dr. H. T. Englehardt has moved from San Antonio to New Orleans, Louisiana.

Dr. Charles W. Evans, Jr., has moved from San Antonio to Del Rio.

Dr. H. D. Giddings has moved from Newgulf to El Campo.

Dr. Manah R. Halbouty has moved from Dallas to Denison.

Dr. J. J. Handley has moved from Fort Bliss to Greenville.

Dr. H. C. Harrell has moved from Texarkana to Fort Sill, Oklahoma.

Dr. E. R. Hayes has moved from Lexington, Kentucky to Greenville.

Dr. A. A. Hellams has moved from Dallas to Ferris.

Dr. L. P. Hightower has moved from Abilene to Fort Worth.

Dr. John M. Hooper has moved from Floydada to Baltimore, Maryland.

Dr. G. R. Howard has moved from Austin to Palestine.

Dr. Hardy A. Kemp has moved from Dallas to Burlington, Vermont.

Dr. Walter L. Kitchens has moved from CCC Camp, Uvalde, to CCC Camp, Marshall.

Dr. J. A. Malone has moved from Beeville to Vanderbilt.

Dr. B. W. Miller has moved from Odessa to Vernon.

Dr. Charles Hyett Miller has moved from Big Springs to Coleman.

Dr. Barth Milligan has moved from Amherst to Austin.

Dr. H. R. Peoples has moved from Harlingen to LaFeria.

Dr. Hal H. Puckett has moved from San Angelo to Texarkana.

Dr. E. C. Price has moved from Quitaque to Bloomfield, New Mexico.

Dr. A. M. Richards has moved from Lone Oak to Centerville.

Dr. Edward S. Stanley has moved from Dallas to San Angelo.

Dr. C. D. Wilson has moved from Temple to Ann Arbor, Michigan.

Dr. T. B. Wilson has moved from Corpus Christi to Bunkie, Louisiana.

Dr. S. L. Witcher has moved from Stephenville to Clifton.

Dr. R. B. Wolford has moved from Mineral Wells to Edinburg.

Dr. Albert M. Gantt has moved from Stockdale to Houston.

Dr. W. A. Grant has moved from Bardwell to San Antonio.

Dr. John E. Manney has moved from Mathis to San Antonio.

Dr. Bert M. Ryan has moved from Dallas to Houston.

Dr. Howard Pleas Wheeler, Georgetown, and Miss Mary Josephine Davidson, Fort Worth, were married August 17.

Continued on page 46

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DRYCO formula modifications, providing moderate fat with ample protein, meet these three primary artificial-feeding needs. In DRYCO feedings, moreover, the protein level, as in breast milk, is *highest during the early months of fastest growth.*

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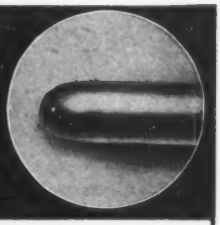

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NOW AT NEW LOW PRICES

Continued from page 44

DEATHS

Dr. Moses Wiesh, San Antonio, aged 85, died August 14 of myocarditis.

Dr. David Forrest Kirkpatrick, Lewisville, aged 78, died August 20 of carcinoma of the bladder.

Dr. George W. Antoine, Houston, aged 60, died August 17 of heart disease.

Dr. Roy Lee Aiguier, Sulphur Springs, aged 50, died September 3.

Dr. Frederick A. York, Navasota, aged 65, died August 22 of paralytic ileus.

Dr. James Henry Burnett, Kopperl, aged 67, died August 22 of hypertension, nephritis and uremia.

Dr. John Davis Duckett, Houston, aged 73, died August 31 of congestive heart disease.

Dr. Ellie H. Putnam, Cuero, aged 73, died August 24.

VIRGINIA

The Fairfax County Medical Society has elected the following officers for the coming year: President, Dr. William Myer, Herndon; Vice-President, Dr. W. P. Caton, Alexandria; Secretary, Dr. Alice H. Kiessling, East Falls Church; Treasurer, Dr. F. M. Brooks, Fairfax Station. The Secretary and Treasurer were re-elected.

The Patrick-Henry Medical Society has elected the following officers for the coming year: President, Dr. W. C. Akers, Stuart; Vice-President, Dr. C. R. Titus, Bassetts; Secretary-Treasurer, Dr. R. H. Walker, Martinsville, re-elected.

The Virginia Urological Society has elected the following officers for the coming year: President, Dr. A. A. Creecy, Newport News; Vice-President, Dr. S. Beverly Cary, Roanoke; Secretary-Treasurer, Dr. Linwood D. Keyser, Roanoke.

The Virginia Radiological Society has elected the following officers for the coming year: President, Dr. Fred M. Hodges, Richmond; Vice-President, Dr. D. D. Talley, Richmond; Secretary-Treasurer, Dr. V. W. Archer, University.

The Virginia Pediatric Society has elected the following officers for the coming year: President, Dr. S. A. Anderson, Jr., Richmond; Vice-President, Dr. J. V. Bickford, Norfolk; Secretary-Treasurer, Dr. J. M. Bishop, Roanoke, re-elected.

Dr. William B. Porter, Richmond, has been elected President of the American Clinical and Climatological Association.

Dr. Ina C. Hall, Richmond, has been granted a leave of absence from his duties as Assistant Director of the Hospital Division of the Medical College of Virginia for a year to do graduate work in Hospital Administration at the University of Chicago.

Dr. I. C. Riggan, Richmond, was recently elected Secretary-Treasurer of the International Association of Medical Health Officers at the Pittsburgh meeting.

The Chesapeake and Ohio Railroad Surgeons have elected the following officers for the coming year: President, Dr. M. J. Payne, Staunton; Vice-President, Dr. Clarence Porter Jones, Newport News; Secretary-Treasurer, Dr. J. M. Emmett, Clifton Forge, re-elected.

Continued on page 48

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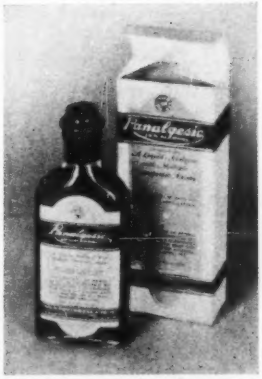

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Continued from page 46

Dr. Charles L. Outland, Richmond, was recently honored by being elected President of the American School Health Association at the meeting in Pittsburgh.

Dr. Sylvester P. Gardner, Derby, was recently honored with a testimonial dinner marking his retirement after twenty-one years on the medical staff of the Stonega Coke and Coal Company.

Dr. Sumter S. Arnim, Richmond, has been appointed Assistant Professor of Operative Dentistry in the Dental School of the Medical College of Virginia.

Dr. David S. Garner, Richmond, Assistant Superintendent of the Hospital Division of the Medical College of Virginia, has resigned to accept the position of Assistant Medical Director of the Shenandoah Life Insurance Company, Roanoke.

Dr. P. R. Cronlund, formerly Assistant Health Officer in Pulaski County, has been appointed to succeed Dr. James R. Suter as Director of the Lee County Health Department. His headquarters will be in Jonesville. During the current academic year Dr. Suter will attend the School of Hygiene and Public Health at Johns Hopkins University.

Dr. E. C. Gates, Bristol, will take a course in Public Health at Johns Hopkins University this year. He has been succeeded by Dr. W. M. Moir as Health Officer for the Washington-Bristol Health Department.

Dr. James T. Rountree has moved from Front Royal to Cora Miller Hospital, Woodstock.

Dr. E. V. Richardson, formerly of Jewell Valley, is now Chief Physician for the Virginia Iron, Coal and Coke Company for the Monarch and Imperial Mines with headquarters at Leona.

Dr. D. H. Rosenfeld announces the opening of offices at 1028 West Franklin Street, Richmond, for the practice of internal medicine and gastroenterology.

Dr. T. B. Washington, Richmond, has moved his offices to the Berkeley Building, 923 West Franklin Street.

Dr. Edwin A. Harper, Lynchburg, announces the removal of his offices to 301 Rivermont Avenue.

Dr. Hugh B. Magill, Jr., has moved from Tappahannock to Gastonia, North Carolina.

Dr. Robley D. Bates, Jr., Richmond, has located in the Professional Building for the practice of internal medicine.

Dr. William D. Tillson, Richmond, Director of the Bureau of Industrial Hygiene of the Virginia State Health Department, has resigned to become Medical Director of the American Viscose Corporation at Parkersburg, West Virginia.

Dr. Donald L. Arey, Danville, and Miss Beverly Jeanne Barclay, Hempstead, New York, were married August 2.

Dr. John Hoge Woolwine, Jr., Blacksburg, and Miss Frances Wellons, South Roanoke, were married September 2.

Dr. Rees Morgan, Roanoke, and Miss Dorothy Hazlewood, Richmond, were married September 30.

Death

Dr. David Finley Brown, Hudgins, aged 82, died August 15 of senility.

WEST VIRGINIA

The House of Delegates of the Medical Society of Virginia has accepted an invitation from the West Virginia State Medical Association to meet jointly at White Sulphur Springs in 1940.

Dr. P. E. Prillaman has moved from Oak Hill to Scarbro.

Dr. J. N. Reeves has moved from Ronceverte to Oak Hill.

Dr. L. I. Hoke has moved from Layland to Nitro.

Dr. W. A. Derrick has moved from Montgomery to Charlottesville.

Dr. R. B. Engle has moved from Longacre to Glen White.

Dr. Peter Darling Crynock, Morgantown, and Miss Louella Wilson, Fairmont, were married November 4.

Dr. William C. Huffmann, Clarksburg, and Miss Henrietta Claxon, Lawrenceburg, Kentucky, were married August 29.

Dr. Peter A. N. Pastore, Bluefield, and Miss Julia Anna Rourke, Prides Crossing, Massachusetts, were married September 14.

Deaths

Dr. Barrick Samuel Rankin, Kingwood, aged 67, was drowned August 22.

Dr. Peyton H. Callway, Beckley, aged 69, died August 1.

Dr. Max William Vieweg, Wheeling, aged 55, died August 13.

Dr. George Holt Barksdale, Charleston, aged 57, died October 9 from a heart attack.

Dr. James Harvey Craft, Springton, aged 65, died September 24.

Dr. Hiram C. Jones, Logan, aged 78, died September 29.

Dr. Etley Price Smith, Fairmont, aged 56, died October 18.

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FOR many centuries,—and apparently down to the present time, even in this country—ricketic children have been passed through a cleft ash tree to cure them of their rickets, and thenceforth a sympathetic relationship was supposed to exist between them and the tree.

Frazer* states that the ordinary mode of effecting the cure is to split a young ash sapling longitudinally for a few feet and pass the child, naked, either three times or three times three through the fissure at sunrise. In the West of England, it is said the passage must be "against the sun." As soon as the ceremony is performed, the tree is bound tightly up and the fissure plastered over with mud or clay. The belief is that just as the cleft in the tree will be healed, so the child's body will be healed, but that if the rift in the tree remains open, the deformity in the child will remain, too, and if the tree were to die, the death of the child would surely follow.

*Frazer, J. G.: The Golden Bough, vol. 1, New York, Macmillan & Co., 1925

It is ironical that the practice of attempting to cure rickets by holding the child in the cleft of an ash tree was associated with the rising of the sun, the light of which we now know is in itself one of Nature's specifics.

New Way . . .

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